The New England Journal of Medicine

Formerly The Boston Medical and Surgical Journal
Established 1828

Volume 219 **SEPTEMBER 22, 1938** **Number 12**

HUMAN ENCEPHALITIS CAUSED BY THE VIRUS OF THE EASTERN VARIETY OF EQUINE ENCEPHALOMYELITIS . . . 411
LeRoy D. Fothergill; John H. Dingle; Sidney Farber and *M. L. Connerley*

THE ROLE OF REST AND EXERCISE IN CONGESTIVE HEART FAILURE . . . 412
David Davis

TWO YEARS OF DIABETIC SURGERY . . . 423
Bernard Rabinovitz and *Jacob Weisman*

RAGWEED-POLLEN SURVEY IN MAINE FOR 1937 . . . 428
Charles B. Sylvester and *O. C. Durham*

DIVERTICULUM AND SARCOMA OF THE DUODENUM . . . 432
Anthony J. Mendillo and *William B. Koufman*

PROGRESS IN DERMATOLOGY . . . 434
Jacob L. Grund

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL . . . 438

EDITORIALS
Organized Medicine Will Meet the Challenge of President Roosevelt's Committee . . . 447
Disgustingly Healthy . . . 447

DEPARTMENTS
Massachusetts Medical Society . . . 448
Miscellany . . . 449
Correspondence . . . 449
Report of Meeting . . . 450
Notices . . . 450
Book Reviews . . . 452

Owned and Published by the Massachusetts Medical Society

Published weekly in Boston at 8 Fenway. **Domestic, $6** per annum, 25c per copy; Canada, $7.04; Foreign, $8.52.
Entered as second-class matter, November 16, 1932, at the post office at Boston, Mass., under the Act of March 3, 1879.

Copyright, 1938, by the Massachusetts Medical Society.

THE MASSACHUSETTS MEDICAL SOCIETY

OFFICERS AND STANDING COMMITTEES ELECTED BY THE COUNCIL, JUNE 1, 1938

PRESIDENT: CHANNING FROTHINGHAM, Boston.
VICE-PRESIDENT: A. WARREN STEARNS, Boston.
SECRETARY: ALEXANDER S. BEGG, Boston.
TREASURER: CHARLES S. BUTLER, Boston.
ORATOR: ELLIOTT P. JOSLIN, Boston.

Executive Offices, 8 Fenway, Boston. Telephone, Ken. 2094.

STANDING COMMITTEES FOR 1938-1939

COMMITTEE ON PUBLICATIONS
R. I. Lee, *chairman;* R. M. Smith, F. H. Lahey, J. P. O'Hare, Conrad Wesselhoeft.

COMMITTEE OF ARRANGEMENTS
R. P. Stetson, *chairman;* Augustus Thorndike, Jr., E. J. O'Brien, W. T. O'Halloran, J. A. Halsted.

COMMITTEE ON ETHICS AND DISCIPLINE
R. L. DeNormandie, *chairman;* C. J. Kickham, R. R. Stratton, W. J. Brickley, A. G. Rice.

COMMITTEE ON MEDICAL EDUCATION AND MEDICAL DIPLOMAS
Reginald Fitz, *chairman;* E. S. Calderwood, A. W. Stearns, A. R. Gardner, G. D. Henderson.

COMMITTEE ON STATE AND NATIONAL LEGISLATION
C. C. Lund, *chairman;* B. F. Conley, A. M. Butler, C. A. Robinson, D. L. Lionberger, *secretary.*

COMMITTEE ON MEMBERSHIP
H. Q. Gallupe, *chairman;* G. C. Caner, J. E. Fish, H. F. Newton, P. Leavitt.

COMMITTEE ON PUBLIC HEALTH
R. B. Osgood, *chairman;* Gerald Hoeffel, S. C. Dalrymple, H. L. Lom F. P. Denny.

COMMITTEE ON MEDICAL DEFENSE
F. G. Balch, *chairman;* E. D. Gardner, F. B. Sweet, A. W. Allen, W. Morrison.

COMMITTEE ON PERMANENT HOME
W. H. Robey, *chairman;* C. G. Mixter, J. M. Birnie, C. S. Butler, E. Miller.

COMMITTEE ON FINANCIAL PLANNING AND BUDGET*
John Homans, *chairman;* E. L. Hunt, W. G. Phippen, C. F. Wilinsky, E. O'Brien.

*Appointed by the President, subsequent to the annual meeting of the Council, as ad interim appointments subject to confirmation at the October meeting of the Cou

OFFICERS OF THE SECTIONS FOR 1939

ELECTED BY THE SECTIONS

(The street addresses may be obtained from the *Annual Directory*)

SECTION OF MEDICINE
Chairman, E. P. Bagg, Holyoke; *secretary*, E. C. Miller, Worcester.

SECTION OF SURGERY
Chairman, B. H. Alton, Worcester; *secretary*, R. H. Smithwick, Boston.

SECTION OF PEDIATRICS
Chairman, E. W. Barron, Malden; *secretary*, J. M. Baty, Belmont and Boston.

SECTION OF OBSTETRICS AND GYNECOLOGY
Chairman, R. J. Heffernan, Boston; *vice-chairman*, M. F. Eades, Newton and Boston; *secretary*, R. S. Titus, Boston.

SECTION OF RADIOLOGY AND PHYSIOTHERAPY
Chairman, H. A. Osgood, Boston; *secretary*, E. C. Vogt, Boston.

SECTION OF DERMATOLOGY AND SYPHILOLOGY
Chairman, E. L. Oliver, Boston; *secretary*, J. G. Downing, Boston.

OFFICERS OF THE DISTRICT MEDICAL SOCIETIES

ELECTED BY THE DISTRICT MEDICAL SOCIETIES AT THEIR ANNUAL MEETINGS, BETWEEN APRIL 15 AND MAY 15, 1938.

(The street addresses may be obtained from the *Annual Directory*)

BARNSTABLE — *President*, C. H. Keene, Chatham; *vice-president*, O. S. Simpson, Falmouth; *secretary*, D. E. Higgins, Cotuit; *treasurer*, H. B. Hart, Yarmouthport; *librarian*, E. E. Hawes, Hyannis.

BERKSHIRE — *President*, H. G. Mellen, Pittsfield; *vice-president*, E. A. Locke, Williamstown; *secretary*, H. J. Downey, Pittsfield; *treasurer*, C. T. Leslie, Pittsfield.

BRISTOL NORTH — *President*, H. L. Rich, Attleboro; *vice-president*, R. M. Chambers, Taunton; *secretary*, W. H. Swift, Taunton; *treasurer*, J. V. Chatigny, Taunton.

BRISTOL SOUTH — *President*, E. P. Seaver, New Bedford; *vice-president*, Thomas Almy, Fall River; *secretary* and *treasurer, pro tempore*, A. H. Sterns, New Bedford.

ESSEX NORTH — *President*, L. R. Chaput, Haverhill; *vice-president*, H. F. Dearborn, Lawrence; *secretary* and *treasurer*, E. S. Bagnall, Groveland.

ESSEX SOUTH — *President*, R. E. Stone, Beverly; *vice-president*, H. A. Boyle, Middleton; *secretary*, J. R. Shaughnessy, Salem; *treasurer*, Andrew Nichols III, Danvers.

FRANKLIN — *President*, F. J. Barnard, Greenfield; *vice-president*, A. H. Wright, Northfield; *secretary* and *treasurer*, Charles Moline, Sunderland.

HAMPDEN — *President*, P. M. Moriarty, Chicopee; *vice-president*, Frederic Hagler, Springfield; *secretary* and *treasurer*, H. L. Smith, Springfield.

HAMPSHIRE — *President*, L. N. Durgin, Amherst; *vice-president*, J. M. Murphy, Northampton; *secretary* and *treasurer*, J. D. Collins, Northampton; *librarian*, Mary P. Snook, Chesterfield.

MIDDLESEX EAST — *President*, R. W. Sheehy, Winchester; *vice-president*, E. Leonard, Melrose; *secretary*, K. L. Maclachlan, Melrose; *treasurer*, Richard Du Wakefield; *librarian*, J. M. Wilcox, Woburn.

MIDDLESEX NORTH — *President*, C. M. Roughan, Lowell; *vice-president*, F. Gage, Lowell; *secretary*, T. A. Stamas, Lowell; *treasurer*, M. D. Bryant, Lo *librarian*, P. J. Meehan, Tewksbury.

MIDDLESEX SOUTH — *President*, F. R. Jouett, Cambridge; *vice-president*, Du O'Hara, Waltham; *secretary*, A. A. Levi, Cambridge; *treasurer*, Edward M Newton; *orator*, R. D. Halloran, Waltham.

NORFOLK — *President*, D. D. Scannell, Jamaica Plain; *vice-president*, C. Kickham, Brookline; *secretary*, F. S. Cruickshank, Brookline; *treasurer*, G. Kaan, Sharon.

NORFOLK SOUTH — *President*, N. R. Pillsbury, South Braintree; *vice-presid* D. B. Reardon, Quincy; *secretary*, R. L. Cook, Quincy; *treasurer*, F. W. Craw Holbrook; *librarian*, R. L. Cook, Quincy.

PLYMOUTH — *President*, B. H. Peirce, South Hanson; *vice-president*, A. W. G Bridgewater; *secretary*, H. C. Reed, Whitman; *treasurer*, A. M. Champ, Brock *librarian*, J. H. Weller, Bridgewater.

SUFFOLK — *President*, Reginald Fitz, Boston; *vice-president*, A. A. Hen Boston; *secretary*, J. P. Monks, Boston; *treasurer*, W. T. S. Thorndike, Boston.

WORCESTER — *President*, C. A. Sparrow, Worcester; *vice-president*, J. C. Au Spencer; *secretary*, G. C. Tully, Worcester; *treasurer*, E. P. Disbrow, Worce *librarian emeritus*, A. C. Getchell, Worcester.

WORCESTER NORTH — *President*, F. P. Moore, Gardner; *vice-president*, B. Sweeney, Leominster; *secretary, pro tempore*, E. A. Adams, Fitchburg; *treas* F. H. Thompson, Jr., Fitchburg.

The New England
Journal of Medicine

Copyright, 1938, by the Massachusetts Medical Society

VOLUME 219 SEPTEMBER 22, 1938 NUMBER 12

HUMAN ENCEPHALITIS CAUSED BY THE VIRUS OF THE EASTERN VARIETY OF EQUINE ENCEPHALOMYELITIS

LEROY D. FOTHERGILL, M.D.,† JOHN H. DINGLE, SC.D.,‡ SIDNEY FARBER, M.D.§ AND M. L. CONNERLEY, M.D.‖

DURING the past summer there has been an epidemic of encephalitis among horses in Massachusetts. In this same period a number of cases of encephalitis have occurred in infants and children. The purpose of the present communication is to announce the isolation, from a fatal human case of encephalitis, of a virus agent which appears to be identical with the Eastern variety of equine encephalomyelitis.

The patient, a seven-year-old boy, was admitted to the Children's Hospital for an illness characterized by an abrupt onset of high fever, generalized rigidity and coma. The patient was extremely ill with a temperature varying between 104° and 105°F. Examination of the cerebrospinal fluid showed 2000 cells with 85 per cent polymorphonuclear leukocytes, a normal concentration of glucose, and a total protein of 190 mg. per cent. Bacteriological cultures were negative. Generalized convulsions occurred and the patient died about twelve hours after admission, forty-eight hours after the onset of his illness.

Postmortem examination of the child revealed acute encephalitis, severe edema and congestion of the brain and cord, generalized visceral congestion, early interstitial pneumonitis and pulmonary edema. The encephalitis was characterized by numerous perivascular accumulations of large mononuclear and polymorphonuclear cells with rare lymphocytes, a diffuse slight infiltration of the meninges by the same cell types, and focal areas of polymorphonuclear and microglial infiltration about dead or injured neurons. These changes were present in many areas of the cortex, the midbrain and the brain stem, and were absent in the spinal cord. Terminal minute thrombi were encountered in the brain and various organs on microscopic examination. No bacteria could be demonstrated in relation either to these thrombi or to the lesions in the brain.

Following necropsy, brain tissue was emulsified in saline and injected intracerebrally into albino Swiss mice. In forty-eight to seventy-two hours these mice died of encephalitis. All bacteriological cultures taken from the human tissue as well as those of mice were negative. In three successive experiments the virus was shown to pass through "N" and "V" Berkefeld filters. Rabbits and guinea pigs were shown to be susceptible to this virus.

An immunologic experiment to identify the virus was carried out as follows: Two guinea pigs,* actively immunized against the Eastern variety of equine encephalomyelitis virus, one immunized against the Western type, and three normal controls were injected intracerebrally with 0.15 cc. of a 1-1000 dilution of mouse-brain virus which had originally been obtained from the brain of the patient. The control animals and the animal immunized against the Western type died of encephalitis in two to five days. The animals immunized against the Eastern type have remained perfectly well. Histologic examination of the brains of mice and guinea pigs which died following inoculation with the virus verified the diagnosis of encephalitis.

SUMMARY

The virus of the Eastern type of equine encephalomyelitis has been isolated from and identified as the etiological agent in a fatal human case of encephalitis. A more detailed report of these immunologic and pathologic studies will be published in the future.

From the Department of Bacteriology and Immunology, the Department of Pathology, and the Department of Pediatrics, Harvard Medical School, and the Infants' and the Children's hospitals, Boston.

†Assistant professor of bacteriology, Harvard Medical School; bacteriologist, the Infants' and the Children's hospitals, Boston.

‡James Jackson Cabot Fellow, Harvard Medical School.

§Assistant professor of pathology, Harvard Medical School; pathologist, the Infants' and the Children's hospitals.

‖Assistant resident in pathology, the Infants' and the Children's hospitals.

*We are deeply indebted to Dr. R. W. G. Wyckoff of the Lederle Laboratories, Pearl River, New York, for his kindness in providing us with actively immunized guinea pigs.

THE ROLE OF REST AND EXERCISE IN CONGESTIVE HEART FAILURE

A Program For Treatment

DAVID DAVIS, M.D.*

BOSTON

IT IS universally appreciated that bed rest is beneficial to patients with congestive heart failure. Except for the use of digitalis in certain conditions, it is the most important and perhaps the only means available of improving a patient with a failing heart. Yet no data are available which might enable the physician to determine how much rest is desirable; and consequently there is no generally accepted plan designed to make the most of this valuable therapeutic agent. In particular there is no agreement as to the length of time the patient should rest after all signs of heart failure have disappeared (Table 1). The vast majority of authors do not specify a given interval, even in the broadest terms. Those who do, usually call for a few weeks at most, and many hold that activity may begin within a few days after evidence of failure has disappeared. Hospital patients are generally permitted out of bed within one to two weeks after an admission for acute congestive heart failure. There is likewise a significant difference of opinion concerning the desirability of graduated exercise in convalescence. Some recommend it strongly, whereas others ignore exercise as though it were of no importance in the treatment of heart disease.

Table 1. *Recommendations Concerning Bed Rest and Exercise in Convalescence Following Cardiac Failure.*

Author	Date	Rest Approved	Importance of Rest Stressed	Definite Period of Bed Rest Recommended	Exercise in Convalescence Recommended
Corvisart[20]	1812	Yes	No	None	
Hope[37]	1839		Yes	None	
Stokes[58]	1854		No	None	Yes
Bamberger[6]	1857	Yes	Yes	None	
Schroetter[52]	1876	Yes	No	None	Yes
Fothergill[29]	1879	Yes	Yes	None	
Flint[27]	1881	Yes	No	None	Yes
Paul[47]	1884	Yes	No	None	Yes
Bramwell[10]	1884	Yes	No	None	
Dujardin-Beaumetz[23]	1887	Yes		None	Yes
Broadbent[12]	1898	Yes	Yes	4-6 weeks	Yes
Balfour[5]	1898	Yes		None	Yes
Gibson[30]	1898	Yes	Yes	None	Yes
Babcock[4]	1903	Yes	Yes	Several weeks	No
Hoffmann[36]	1905	Yes	Yes	None	
Satterthwaite[51]	1905	Yes	No	None	Yes
Colbeck[18]	1905	Yes	Yes	None	Yes
Brunton[15]	1908	Yes	Yes	None	Yes
Krehl[41]	1908	Yes	Yes	None	Yes
Jürgensen[40]	1908	Yes	Yes	None	Yes
Hirschfelder[35]	1910	Yes	Yes	None	Yes
Osborne[48]	1916	Yes	Yes	None	Yes
Mackenzie[44]	1916	Yes	Yes	None	Yes
Neuhof[45]	1917	Yes	Yes	2-3 weeks	
Wilson[62]	1918	Yes		None	Yes
Pratt[49]	1920	Yes	Yes	4-6 weeks	No
Greer[31]	1922	Yes	Yes	None	Yes
Coombs[19]	1924	Yes	Yes	2-4 weeks	
Wynn[62]	1925	Yes	Yes	None	
Hunt[38]	1925	Yes		None	Yes
East and Bain[24]	1929	Yes	Yes	None	Yes
Romberg[50]	1929	Yes		None	Yes
Heimann[34]	1930	Yes	Yes	None	
Brams[9]	1931	Yes	Yes	None	
Bramwell[11]	1933	Yes	No	None	
Lewis[43]	1933	Yes	Yes	12-24 weeks	
Harrison, Friedman, Resnik and Clark[32]	1934	Yes	Yes	None	
Boas[5]	1934	Yes	Yes	4-12 weeks	
Brooks[13]	1934	Yes	Yes	None	Yes
Siebeck[56]	1934	Yes	Yes	Several weeks	
Scherf[54]	1935	Yes	Yes	Several weeks	
Jagic and Flaum[39]	1935	Yes	Yes	None	
Christian[16]	1935	Yes	Yes	Several weeks	
Warfield[60]	1935	Yes	Yes	None	
Eggleston[26]	1935	Yes	Yes	1 week	Yes
Paullin and Minnich[46]	1936	Yes	No	None	
Strauss[59]	1936	Yes	No	None	
Levine[42]	1936	Yes	Yes	None	No
White[63]	1937	Yes	Yes	Few days to few weeks	Yes

An historical review of the role played by rest and exercise in the treatment of heart disease suggests that much of our present-day confusion is a heritage of the past. The value of rest in heart

*Assistant in medicine, Harvard Medical School; assistant physician, Beth Israel Hospital.

failure was hardly recognized until the latter part of the nineteenth century. On the other hand, the use of exercise long antedated the use of rest in the treatment of a weakened myocardium. In spite of the recommendations of Hope,[37] Bamberger,[6] Fothergill,[28, 29] Sibson,[55] Balfour[5] and Hasebroek,[33] exercise continued to hold precedence over rest, and in the eighties and nineties the Nauheim treatment of baths and exercises was almost universally accepted. Since then there has been a gradual movement away from this tenet, but the influence of these earlier teachings is still to be observed in our present ideas concerning the value of these therapeutic agents. It is probable that the vagueness of our position concerning the amount of rest desirable for patients with heart failure stems from an inability to reconcile the implications of rest with the paradoxical claims for exercise.

In the present study the following aspects of the problem will be considered: the historical development of the use of rest and of exercise in heart disease; a critique of the use of exercise as a therapeutic agent; a comparison of the results of prolonged rest in one group of patients with the results after shorter periods in another group with congestive heart failure; and the development of a comprehensive program of treatment based on available data.

EARLIER VIEWS ON THE ROLE OF REST AND EXERCISE IN HEART DISEASE

Senac's[53] treatise in 1749, the first dealing with heart disease, had little to say concerning the role of rest or exercise. Apparently one of the first comments on the subject was made by Corvisart[20] in 1812 when he spoke of the harmful effects of exercise. Referring unmistakably to heart failure he said:

> . . . but it seldom takes its natural course; its progress is commonly hurried by the mistakes in regimen, in exercise and by the moral affections, while by observing a proper regimen, abstaining from all violent exercise, and fatiguing employments with tranquility of mind, persons attacked with the organic lesions of the heart might some time exceed the mean term among the probabilities of human life.

Hope[37] was probably the first to stress the importance of rest. In 1839 he stated that satisfactory results in chronic endocarditis could be obtained by "confining the patient to bed for the purpose of assuring the utmost possible corporeal tranquility. . . ." Concerning exercise he added: "Any exercise taken should be so gentle as never to hurry, and, if possible, not even to accelerate the circulation beyond a few beats. Walking uphill is, therefore, out of question, *even though the patient declare that he can do it without inconvenience. . . .*" In the light of later trends this recognition of the importance of having a cardiac patient who has had failure do less than he could without discomfort was most unusual. Bamberger,[6] writing in 1857, likewise recommended absolute bed rest as the best form of treatment in rheumatic heart disease and, like Hope, cautioned strongly against overexertion.

In 1854 Stokes[58] published his monograph on heart disease. Referring to so-called fatty degeneration of the heart, he wrote:

> We must train the patient gradually but steadily to the giving up of all luxurious habits. He must adopt early hours, and pursue a system of graduated muscular exercise; and it will often happen that after perseverance in this system the patient will be enabled to take an amount of exercise with pleasure and advantage, which at first was totally impossible, owing to the difficulty of breathing which followed exertion. . . .

There is no reason to believe that these recommendations were meant for any other form of heart disease. Nevertheless they were taken to apply to other forms of heart disease and this influence prevailed. In the ensuing years not rest, but exercise was the major theme. One of the strongest advocates of this regimen for cardiacs was the famous American clinician, Austin Flint.[27] When cardiac symptoms have led the patient to the physician, he wrote in 1859:

> Moderate open air exercise is to be encouraged, very active exertion being, of course, interdicted, . . . patients exchanging habits of activity for complete rest are likely to become rapidly worse. . . . If dilatation predominate, the measures indicated are still those which tend to give, as far as possible, tone and vigor to the heart. . . . The diet is to be highly nutritious, although as dry as is compatible with comfort; tonic remedies are to be employed, and moderate exercise out of doors is still advisable.

Flint believed that rest weakened the heart and that exercise could strengthen it.

With the exceptions noted there were few writers prior to 1872 who expressed a full appreciation of the value of rest. In this year Fothergill[28] first published his treatise on heart disease, and in it maintained that bed rest gave relief in every case of heart failure due to myocardial weakness.

> One of the most unfortunate scepticisms in relation to diseases of the heart arises from the often wonderful effects of rest. This is mostly seen in hospitals. A patient with dyspnea, cyanosis, feeble irregular pulse, and small bulk of urine passed in the twenty-four hours, is taken in and put into bed by the house surgeon, or clinical clerks, in the presence of a class of students. No treatment is decided upon until the case is seen by a physician, and in the meantime the patient improves very much, his cough is less troublesome, his respiration less impeded, he can breathe lying down, or with much less propping, and the pulse becomes much steadier. This improvement is so

striking, that "the expectant treatment" is continued until the patient is sufficiently recovered to resume his occupation. The case creates comment, and illustrates beautifully the effect of rest as part of the treatment of heart disease, leaving the impression on many minds that rest in bed is the treatment, *par excellence,* for heart failure. But follow the patient a little longer, and see the effect of the resumption of exertion, the toil necessary to procure the daily bread, and you would see every symptom return with its former or increased force. The heart is quite equal to the call upon it when lying in bed, and even to somewhat of compensatory growth under these favorable circumstances, but the resumption of toil soon brings out its incapacity.

About this time Sibson[55] was making a statistical study of the results of bed rest in patients with rheumatic fever. Patients treated during a period of 15 years, from 1851 to 1866, were not kept in bed; and the results in this group were compared with a second series in which bed rest was employed throughout the attack of rheumatic fever. Of the patients who were allowed up and about the wards, over twice as many subsequently developed severe heart disease. This work, as Pratt[49] noted, was of fundamental importance, but it marked only the beginning. Sibson had treated rheumatic fever, not rheumatic heart disease, and after symptoms of arthritis disappeared, rest was not prescribed even if significant cardiac damage had developed. Fothergill, who recognized the importance of Sibson's work, also appreciated that it did not go far enough, and in the second edition of his book,[29] published in 1879, he stressed the need of rest in acute endocarditis and other forms of cardiac weakness.

Balfour[5] likewise showed an appreciation of the importance of rest, and with Fothergill advocated it when others in Europe were insisting on as much exercise as could be taken without discomfort:

> Concerning the difficulty most cardiac patients have in making any exertion, it seems almost superfluous to insist upon the importance of rest, yet it is a matter which cannot be too much insisted upon. . . . Whenever, then, from any cause there is defective, irregular or in any respect uncomfortable action of the heart, and still more whenever we have any uncompensated or imperfectly compensated valvular lesion, rest must be absolutely insisted upon as a necessary adjunct to the treatment, and it must be maintained for a few days or a few weeks until the heart acquires a certain amount of pristine vigor. In very many cases the mere act of getting out of bed and lying on a sofa for a few hours is too much for the patient and delays his recovery considerably; while any greater exertion not infrequently thwarts all our endeavors to benefit the patient, and at all events renders our treatment less safe and certain. You must never forget, therefore, to insist upon rest as a most important part of the treatment, a rest which must be the more perfect and complete the more serious the cardiac disturbance is.

Fothergill did not specify how long rest should continue, and it is noted that Balfour speaks of only a few days or a few weeks of rest at most. Balfour likewise had some faith in the possibilities of exercise:

> Exercise and diet are, however, paramount in maintaining the integrity of a healthy organism; and properly employed, they are also of the greatest value in restoring it when lost.
>
> It seems somewhat of a paradox to speak of exercise as a treatment for an organ which takes its needful rest in sections, and that only for a fraction of a minute, and which, as a whole, is in a constant and continuous work from man's birth to his death.

Balfour then goes on to say that like any other muscle the heart is probably strengthened by exertion and that exercise judiciously employed may be profitable. This analogy with skeletal muscle probably played an important part in holding out favorable expectation from a regime of exercise.

From this time on, through the beginning of the twentieth century, one finds an increasing appreciation of the value of rest with less and less emphasis on exercise. Few authors, however, dispense entirely with the aid of exercise, and only occasionally is a desirable period of rest specified. Broadbent,[12] writing in 1898, was one of the first to say just how long rest should be continued. "Rest is in many cases an important part of the treatment, and when the state of the patient demands it, rest in bed is of the greatest service; but one of the questions calling for the greatest exercise of judgment is to decide when absolute rest is necessary. There are circumstances in which a month or six weeks in bed will prolong life for as many years." But like his contemporaries, Broadbent was not entirely committed to rest and not yet ready to strike out against exercise. "Even when severe symptoms are present, to insist upon confinement indoors is sometimes to add to the suffering of the last few months of life without adding to its duration." He recommended the exercise treatment of Oertel, although not very enthusiastically. In rheumatic endocarditis, however, he made an unusual recommendation, namely "weeks or months" of bed rest as a *prophylactic.*

In the next decade or two opinion swerved farther to the side of rest, but many outstanding clinicians still continued to talk of the importance of exercise as soon as symptoms of failure had disappeared. Thus Colbeck,[18] Brunton,[15] Krehl,[41] Hirschfelder,[35] Osborne,[46] Mackenzie[44] and Allbutt,[1, 2] clinicians who wrote eloquently of the value of rest, nevertheless also stressed the importance of exercise in convalescence. Brunton, who advised that patients "shall not take one beat out of the heart that can possibly be avoided," recommended the Oertel treatment in convalescence and gentle exercises in cardiac asthma "to train the heart to increase its nutrition and accelerate the

circulation through the lungs." Osborne[46] presented a similar thesis, as did Mackenzie, in spite of the fact that the latter undertook a study of the Nauheim treatment and came to the conclusion that the benefits were largely mental. Mackenzie writes:

> When exhaustion has gone so far that breathlessness or pain is easily induced, and objective signs, such as dropsy, are present, rest is imperative. While other remedies may be necessary, rest is the most potent remedy of all. When the patient is fit to go about he should be encouraged to take exercise in the fresh air. The amount of exercise he may take will depend upon his strength and this strength can be gauged by his sensations. So long as a man who is taking exercise remains free from distress or discomfort, then the exercise is doing him good.

Contrast this with the advice of Hope,[37] who urged patients to do less than what they could do in comfort.

Allbutt's[1, 2] recommendations were likewise somewhat contradictory. Referring to rest he writes:

> But, however, apparently imperative and universal, the insight of Stokes perceived that this rule needed much modification. In hopeful cases as we shall see hereafter, to make the heart a permanent invalid is as bad a general rule as permanently to invalid the body.
>
> The manner in which Oertel at first prescribed hill work to his patients, or how Zanider and August Schott prescribed more artificial gymnastics, is, save as a matter of history, of secondary importance; now we are all agreed that whatever nicety may be required in working out the comparative values of the means themselves, in heart disease, the rule of continuous bodily repose needs large modifications.

One gets the impression from Allbutt for a moment, that Stokes had not gone far enough.

In 1920 Dr. Joseph H. Pratt,[49] of Boston, published an interesting paper dealing at length with this aspect of the problem. He exposed the inconsistencies and inadequacy of present-day teaching and more than any other clinician recommended more prolonged rest in the treatment of congestive heart failure.

Table 1, arranged chronologically, summarizes the views of a representative group of past and present-day clinicians. It will be seen that from 1900 to 1930 the vast majority recommended both rest and exercise. During the past decade fewer have stressed exercise in convalescence, but the number who commit themselves as to how long rest should continue is small. It will be noted, for example, that outstanding clinicians recommend as little as one week of bed rest after evidences of heart failure have disappeared. An exception is Sir Thomas Lewis,[43] who states that six to eight weeks of bed rest is rarely sufficient and that three to six months is desirable.

THE ROLE OF EXERCISE

The analogy to skeletal muscle is partly responsible for the tenacity with which physicians adhere to the idea that exercise may improve a weakened myocardium. It is known that the strength of skeletal muscle may be increased by training and weakened by disuse. Heart muscle, as others have pointed out in this connection, has a structure and function that is in some respects decidedly different from that of skeletal muscle. A more serious objection, however, to the analogy is the fact that the response of a healthy structure is compared with the response of one that is diseased.

An analysis of what occurs in the course of failure due to arteriosclerotic heart disease (hypertensive, rheumatic, luetic and congenital heart disease are ruled out) throws some light on the limitations of exercise in this disease. In a recent study it was pointed out that failure in this etiologic group is due primarily to disease of the coronary vessels.[22] An increase in cardiac work in the presence of some coronary disease generally leads to further impairment of nutrition. This follows because an increase in work is accompanied by an increase in cardiac output and this demands a corresponding increase in coronary circulation.[57] If the coronary tree is already so diseased that it cannot accommodate the normal requirements of the heart muscle, it obviously will be unable to satisfy the requirement under the conditions of exercise and further damage to the structure is inevitable. It is impossible, of course, to determine at any moment what are the exact needs of the myocardium in respect to coronary artery capacity. The chances of the requirements exceeding the coronary capacity during exercise in convalescence, however, must be great in patients just recovering from heart failure initiated by an inadequate coronary circulation in the first instance. Just how exercise could improve the coronary circulation under such circumstances is certainly not clear.

It is also difficult to understand how exercise can improve the situation when heart failure is due to hypertensive or valvular heart disease. Hypertensive hearts are characterized by consistent hypertrophy of the muscle fiber. Wearn[61] has recently pointed out that the hypertrophied heart is less efficient for the reason that its capillary blood supply is relatively reduced. A situation of this kind would tend to be aggravated rather than improved by exercise for the same reasons mentioned in connection with coronary heart disease.

For the same reasons it is difficult to understand how exercise, even within limits, would be beneficial in other diseases of the myocardium.

The support for a therapeutic agent, however

must rest mostly on the results of its application. If exercise within certain limits improves a weak myocardium there should be data in the literature to support it. Such studies purporting to demonstrate improvement either fail to do so or appear to deal with conditions other than heart failure. Some of these are the following: obesity with dyspnea on exertion; compensated heart disease, such as rheumatic heart disease before failure has occurred; and psychoneurosis with symptoms suggesting circulatory insufficiency.

Hasebroek[33] pointed out that what Oertel in his own case took as a demonstration of improved cardiac function was the best evidence in the world that Oertel did not suffer from heart disease. His capacity to climb appreciable mountain heights in a short period of time was more than could be expected from untrained skeletal muscle. Hasebroek noted that 27 of Oertel's 51 cases were merely obese. One gets the impression that Oertel did not have a clear conception of what constituted heart failure; and it appears that he treated obese, compensated cardiacs and psychoneurotic patients with the mistaken idea that they were suffering from a weak myocardium.

It is well known that psychoneurotic patients commonly complain of symptoms which suggest circulatory insufficiency. Also patients with compensated heart disease of rheumatic or congenital origin long aware of their condition may go through life with a mild neurosis, complaining of slight dyspnea on exertion and other symptoms which in the course of years do not show any progression. One can understand, under such circumstances, how an exercise regime which preoccupies the mind may result in the disappearance of symptoms, at least for a time. Daly and Strouse,[64] for example, maintained that patients with rheumatic heart disease in the course of pregnancy do better or feel better, when they are permitted more activity than is customarily recommended. Strouse has maintained that cardiac patients do not do well with too much rest. His cases, however, were compensated and improvement could not have been related to the cardiac status.

There is another sequence of events which suggests, at first, that too much rest may not be good for cardiac patients. When older patients are let out of bed after weeks of confinement because of some medical or surgical condition symptoms are occasionally observed which suggest circulatory insufficiency. It is noticed at times that there is dyspnea on slight exertion, slight pitting edema of the legs and rarely rales at the lung bases. These signs are apt to be attributed erroneously to cardiac disease acquired as a result of prolonged bed rest. As one observes these patients many weeks further, however, it is seen that the dyspnea, edema and rales eventually disappear in spite of slowly increasing activity. This is similar to the sequence of events that occurs when a young individual with a normal heart is confined to bed for several weeks. Dyspnea and weakness are notable on the slightest attempt to increase activity over a certain limit, and only after weeks of gradually increasing activity is there a return to the previous capacity. This adjustment may be partly related to adjustments in coronary circulation, which must attend the increase in cardiac work. Elderly patients with a narrowed coronary bed probably require more time to make the adjustment, and if heart function is taxed excessively before this occurs, symptoms of cardiac weakness may appear, at least for a short time.

If exercise in convalescence after congestive failure is beneficial then rest could not be, for obviously rest and exercise at any one time are diametrically opposing forces. Those who believe exercise is beneficial or that too much rest is harmful, however, usually maintain that this only applies when symptoms of failure have subsided. Increased activity is held desirable only when the heart has presumably improved, and the signal for such improvement is generally taken as some time shortly after the disappearance of signs and symptoms. In order to obtain a clearer meaning of this, let us consider what occurs in the course of recovery. As a result of rest the reserve increases to a point at which it can take care of the circulatory requirements under almost basal conditions and discharge any previously accumulated debt. At this point the usual signs and symptoms have disappeared, but the reserve is often far below the level that will be achieved if the recovery process is unimpeded. The degree of possible improvement beyond this point will obviously vary in different patients, and the time necessary to reach this optimum will also vary. In any case, if patients are permitted activity before it is reached the benefits of rest must necessarily be vitiated.

RESULTS IN A GROUP OF HOSPITAL PATIENTS WITH CONGESTIVE FAILURE COMPARED WITH THOSE TREATED IN PRIVATE PRACTICE ACCORDING TO A PROGRAM OF PROLONGED REST

In order to determine the importance of prolonged rest, its results in a group of cases might be compared with those treated in the larger hospitals, a regime which generally involves much less initial rest and subsequent reduction in activity. For controls the course in 50 unselected patients with hospital admissions for congestive heart failure who survived the first attack was analyzed. Their course is plotted in Figure 1. Hospital admissions with congestive failure are indicated by

dark, erect bars. The thin line connecting erect bars represents a period of freedom from signs of congestive failure, although the patient may have complained of mild dyspnea on exertion, palpitation, precordial pain or infrequent attacks of cardiac asthma during this time. The double line indicates complete freedom from evidence of failure. A study of this figure shows that the longest interval between the first and second attacks of failure was three and a half years. This should be compared with the findings in the group receiving prolonged bed rest, as indicated in Figure 2. Here it will be noted that 7 out of 11 cases were free of evidence of congestive heart failure for three or more years, and that 3 have thus far maintained this good health for a period of five or more years.

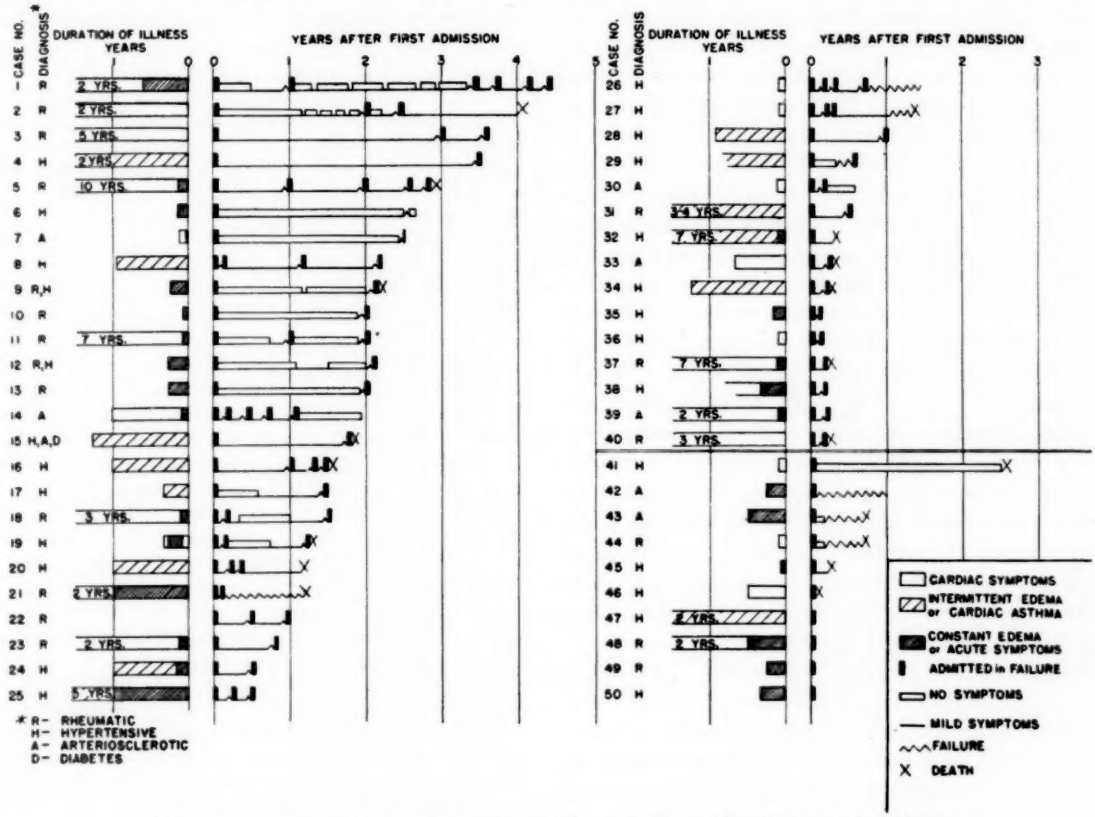

Figure 1. *Course after congestive heart failure in 50 unselected hospital patients.*

Forty of the patients in the control groups had a subsequent hospital admission, as indicated in Figure 1. The remaining 10 patients had only one admission. The subsequent course was ascertained in 6 of these. In 4 no follow-up data could be obtained. The course of this group of 46 unselected patients (Figure 1) is essentially the same as that found in two other Boston hospitals whose

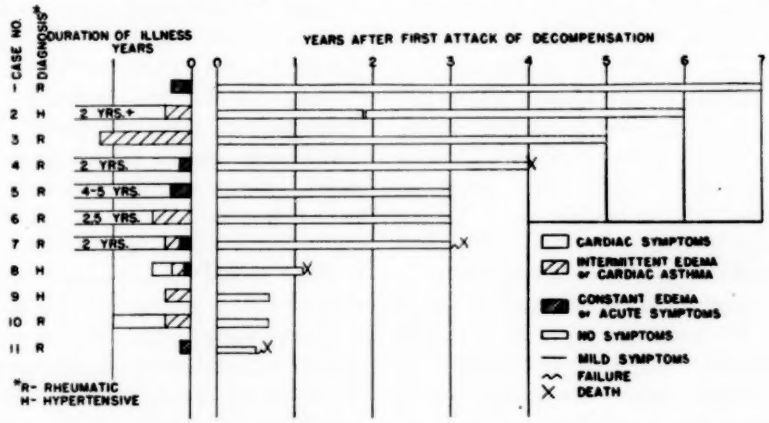

Figure 2. *Clinical course after congestive heart failure in 11 patients treated with prolonged bed rest (three to twelve months).*

records were available, and is in conformity with similar data in the literature concerning the course of patients after the beginning of congestive heart failure.[21] It will be noted that 30 of the group of 40 with two or more hospital admissions had a second admission for congestive failure within one year after the first, 21 within six months and 15 within three months after they were first discharged. Several of the latter had to be readmitted within two to four weeks. The course in 6 of the 10 cases with but one admission was essentially the same. In many cases the first hospital admission was less than two weeks, and in all cases patients were permitted the use of a chair and lavatory at least two or three days before discharge. In approximately 50 per cent of cases further bed rest in a convalescent home or at home was recommended, but this was rarely for more than two to four weeks. Absolute bed rest without lavatory and table privileges was seldom recommended and more rarely carried out. Patients were usually discharged within a week or two after symptoms of failure had disappeared, and a few weeks of bed rest at home was recommended. The total rest period was rarely more than two months, even after severe decompensation.

The group of patients summarized in Figure 2 followed a program consisting of a long initial period of absolute bed rest related to the severity of the illness and varying from three months to one year after symptoms of failure had disappeared in the 11 cases studied, a subsequent reduction in activity related to the level of activity prior to the onset of the illness and the severity of the recent illness, and periodic prophylactic bed rest. Although the number studied in this way is small, the differences are so striking as to leave little doubt as to the efficacy of such a program. In every way these patients were comparable to the control group. They came for treatment and were hospitalized for congestive failure before the rest program was outlined. Case 1 has gone seven and a half years, Case 2 six years and Case 3 five and a half years since the first onset of failure. Case 4 was first seen five years before death, complaining of severe dyspnea. Physical examination showed no objective evidence of failure, but the electrocardiogram showed marked bundle-branch block. One year later the patient developed an attack of marked congestive failure. She improved strikingly after a six-month initial rest period and thereafter showed no evidence of failure under a regime as outlined above. Death four years after the onset of failure was sudden; it was preceded by attacks of syncope but was unaccompanied by evidence of failure. Case 8 also had bundle-branch block and death was sudden and unaccompanied by congestive failure.

In a recent study of the pathologic physiology of chronic cardiac decompensation Altschule[3] points out that at the time of discharge from the hospital patients with congestive heart failure are often in a condition of comparative failure, in spite of the absence of the usual signs. Altschule summarizes his studies as follows:

> A group of patients were studied in order to determine the physiological status of the patient with chronic cardiac decompensation in whom the greatest possible improvement in symptoms was secured by treatment in the hospital. . . . These patients received the benefit of complete bed rest for several weeks, limitation of fluids and salt, full doses of digitalis, adequate diuresis with mercurial preparations, and in a few instances venesection or oxygen therapy as well. It is evident that striking symptomatic improvement had occurred; that is, there was no edema and at most minimal dyspnea at rest. However, evidence of the persistence of an abnormal cardiovascular physiology was shown by the somewhat decreased cardiac output, tendency toward an increased respiratory rate and minute volume, a slight decrease in arterial blood oxygen saturation, a lowering of the vital capacity, a prolongation of the pulmonary circulation time, and, in one patient, slight elevation of the venous pressure. These findings corroborate and extend the observations of other authors who have studied the cardiovascular system after recovery from congestive failure. The persistence of abnormal physical findings such as cyanosis, cardiac enlargement, and hepatic enlargement in the cases here reported is to be noted. The symptoms of dyspnea, orthopnea and edema were controlled but the cardiovascular physiological measurements in each case failed to return to normal values. Following discharge from the hospital all the patients, except one who died, returned with fully developed cardiac decompensation within a year, in spite of the fact that their activities were markedly limited.

The view might be taken that the group of patients who had prolonged bed rest in contrast to the hospital group did better because their subsequent activities were better controlled and not because the initial rest period was long. It is true that the subsequent activity in the group receiving the long initial period of rest was carefully controlled. In the last analysis, however, most of these patients were permitted degrees of activity comparable to that found in the majority of the control group. For example, the patient in Case 1 has worked as a newspaper clerk for seven and a half years. He stands approximately four hours a day and makes up newspaper bundles for distribution. The patient in Case 2 is out-of-doors five or six days a week. She goes shopping, visits friends and takes in all amusements. These activities entail some walking, although this is reduced to a minimum. The patient in Case 3 began cooking one meal a day one year after the onset of treatment and has continued this for five years. The patient in Case 7 worked as a saleslady the first year after the onset of treatment. She then married and for two years thereafter did her own cooking. Only 1 patient (Case 5) in

this group has lived a chair-and-bed existence. On the other hand, many of the hospital patients redeveloped failure shortly after leaving the hospital and before their activity had become appreciably increased, thus indicating unmistakably that the initial period of rest was inadequate.

A PROGRAM OF REST TREATMENT IN HEART FAILURE

Although digitalis was first introduced by Withering, in 1785, its clinical use was not generally appreciated until the present-day method of administration was proposed two decades ago. The therapeutic status of rest today is similar to that of digitalis before the twentieth century. As pointed out above, the paradoxical claims for exercise and the notion that rest in itself may weaken a diseased myocardium have hampered the development of a logical program. Such a program has at least three important considerations: the duration of the initial period of absolute bed rest, the degree and character of reduced activity that must follow and the advisability of periodic prophylactic bed rest.

The Duration of Bed Rest

The duration of bed rest should obviously be related to the severity of the circulatory insufficiency. In practice this has generally meant that the patient with a severe condition, whose signs and symptoms of failure cleared slowly, was forced to remain in bed for a longer period than one less severely ill. Statements such as "The patient has been in bed now for ten weeks and that is enough if he is to get well at all" are commonly heard. The more important question, "How long has the patient been free of evidence of failure?" is generally neglected. The difference between being in bed ten weeks with the last nine free of signs of failure and ten weeks with the last one free of failure is considerable.

The time required for evidence of failure to disappear under a regime of absolute bed rest is a means of roughly measuring the severity of the underlying condition and the cardiac reserve. Having estimated the cardiac reserve in this manner, we can arrive at a desirable period of additional bed rest on the basis of certain theoretical considerations and the results of the above experience. For working purposes, we may consider two stages of improvement after evidences of failure have disappeared: first, that in which the cardiac reserve still continues to increase in the course of days or weeks to a certain optimum; second, a period after this in which the gain in reserve as measured by capacity to perform a given task no longer shows very much change but in which the potential capacity of the heart is being enhanced. This change is probably in the nature of organic repair, such as may occur after rheumatic carditis or failure due to coronary sclerosis. In coronary disease, for example, improvement may be related to the better nutrition that follows the development of a more adequate anastomotic circulation.

What happens in this first stage of rest is well recognized. At the point where evidences of failure disappear a patient may be a long way from the recovery level he is potentially able to attain, as evidenced by a low cardiac output, a slow velocity of circulation and the inability to carry on any appreciable effort without provoking symptoms. As rest continues the cardiac reserve approaches a higher level, as demonstrated by the ability of the patient at this time to perform certain physical tasks without producing an abnormal oxygen debt. For example, a patient shows progressive dyspnea on exertion for a period of months and then develops acute pulmonary edema. After a few days in bed signs in both lung fields become very much improved and subjectively he is free of symptoms. In the course of another four or five days only a few rales at the lung bases are audible, and after ten to fourteen days in bed there is no evidence of congestive failure. If recovery continues to progress at the same rate, it is reasonable to believe that two or three weeks must ensue before a certain optimum capacity is established. But after this level has been achieved there is the second phase of recovery to consider. Recovery in this stage, as mentioned, may not be measurable in terms of an increased capacity to perform a given task but rather as an increase in the potential capacity, an increased factor of safety. In the case under consideration approximately four weeks of bed rest will probably be necessary before the optimum level is reached. But since the second period of recovery is far more significant in terms of the future, the rest phase should be at least twice and preferably three times that required for the first stage. In this case, then, the patient should remain in bed from twelve to sixteen weeks after the onset of failure.

Table 2. *Requirements of Bed Rest in Relation to Duration of Cardiac Failure.*

Duration of Cardiac Failure	Therapeutic Bed Rest		Prophylactic Bed Rest with Bathroom and Table Privileges
	Minimal	Desirable	
wk.	*wk.*	*wk.*	*wk.*
2 or less	12	16	1 out of 8
4	16	24	1 out of 6
8	32	48	1 out of 4
12	48	72	1 out of 3

Table 2 gives an outline of the probable requirements of bed rest in the light of the above considerations.

Reduction in Activity

Some reduction in activity is unquestionably desirable after the proposed initial period of bed rest has come to an end. The degree will naturally be related to the severity of the recent illness and the degree and character of activity preceding the onset of failure. The longer the initial period of bed rest the greater the relative reduction in activity. In many cases the reduction may still permit engagement in some useful but less strenuous occupation. A housewife's activity may be reduced to cooking but one meal a day, all other activities, including stair climbing and walking, except for such essential steps as from house to car or car to theater seat, being prohibited.

In the case of individuals who have had a sedentary occupation, it is necessary to focus all attention on the non-working hours. The desired effect may be accomplished here by appreciably increasing the sleep and bed-rest hours, eliminating all stair climbing and walking, and spending the greater part of week ends in bed. Frequent periods of prophylactic bed rest are especially desirable in such patients.

Prophylactic Bed Rest

Prophylactic bed rest is an important part of the program in the treatment of patients with cardiac failure. The ultimate benefits to be obtained from rest are obviously greater when it acts to prevent failure rather than after failure has appeared. Since it is a fact that patients who have had failure sooner or later show a recurrence after recovery, prophylactic periods of bed rest in such patients become of paramount importance. The amount of such rest will naturally vary with the severity of the illness. Table 2 gives a rough estimation of what this might be. Bathroom and table privileges may be granted, but no others.

SUMMARY AND CONCLUSIONS

There is no agreement in the literature as to the length of time patients should rest after evidence of congestive failure has disappeared, or as to the advisability of exercise in convalescence. The historical aspect of these questions is considered.

The question of the advisability of exercise in convalescence is discussed at length. No data were found to support the contention that this therapy improves the capacity of the heart.

The results of prolonged bed rest in 11 cases are compared with those in a control group treated in the accepted manner. The course in these 11 cases was appreciably better than that in the control group.

These results call for a program which will take into consideration an adequate initial period of rest, adequate subsequent reduction in activity and periodic prophylactic bed rest. Such a program is outlined.

SAMPLE CASE REPORTS

Case 1. T. S., male, aged 27. Rheumatic heart disease, mitral stenosis, auricular fibrillation, congestive heart failure; dyspnea 3½ months. Course 7½ years after beginning treatment.

This patient was first seen December 29, 1930, complaining of progressively increasing dyspnea on exertion of 3½ months' duration. The dyspnea was accompanied by cough productive of frothy sputum and often occurred in the form of severe paroxysms lasting 10 minutes. He was forced to give up work entirely during this period. He consulted several physicians who recommended rest, but not too strongly, and prescribed varying amounts of digitalis. In spite of this his condition became progressively worse. He had had chorea at the age of 14, one sister had chorea at the age of 10, and his father had apparently died of rheumatic heart disease at an early age. Physical examination showed good development, slight cardiac enlargement, a grossly irregular rhythm, a ventricular rate of 120 and a rough early and mid-diastolic murmur at the apex. The blood pressure was 110/70 mm. There were a few rales at the lung bases. The liver edge was not felt and there was no edema.

The patient was put to bed in a hospital and more fully digitalized. The ventricular rate slowed to approximately 90. The lungs became clear. The white-cell count and sedimentation rate were well within normal limits. After 2 weeks of absolute bed rest quinidine was administered and the auricular fibrillation was converted to normal sinus rhythm, with rates varying from 90 to 100. Absolute bed rest was continued for 10 weeks further, and 2 or 3 weeks after this he returned to his job as a mailing clerk in a newspaper office. His work called for standing for approximately 4 hours of the 8-hour day and at times the lifting of packages weighing as much as 75 lb. Arrangements were made so that he did not have to lift such weights very often, and a plan was outlined so that activity in his leisure hours could be reduced to a minimum. He was driven to and from work and agreed not to walk an extra step beyond the minimum required by his work. He spent 12 to 14 hours daily and the greater part of the 24 hours on Sunday sleeping or in bed. Whenever he could obtain time off he spent a week in bed as a prophylactic measure. In May, 1934, the patient observed that his heart action had become rapid and irregular. Physical examination showed auricular fibrillation. He was given quinidine and the rhythm was converted to normal. He rested in bed as a prophylactic measure for 3 weeks and resumed work 1 week later. In October, 1936, there was a recurrence of rapid and irregular heart action. Physical examination showed auricular fibrillation, rate 160. Quinidine was prescribed and the rhythm was restored to normal for the third time. The patient rested in bed for 2 weeks before resuming work. In September, 1937, auricular fibrillation recurred for the fourth time. Quinidine again restored the rhythm to normal in the same manner. He stayed in bed for 2 weeks and then resumed work.

At the time of writing, June, 1938, this patient was still at work. Throughout the period of 7½ years he had kept to the prescribed regime concerning reduction in leisure-time activity and prophylactic bed rest. Two years after the onset of his illness he was married. Except for the four attacks of auricular fibrillation that responded promptly to quinidine therapy he had had no cardiac illness since rest treatment was first instituted. Digitalis was not administered except at the onset of his illness, before quinidine

therapy. After normal rhythm was restored, digitalis was omitted and not resumed. He was seen for routine check-up examination approximately once a month. The pulse rate varied from 90 to 100. Repeated temperature readings, white-cell counts and sedimentation rates were always normal.

Comment. When he first began to show evidence of congestive failure, it is probable that his failure was due primarily to the abnormal rhythm and the rapid ventricular rate. As the rates, however, were not unusually high it is likely that there was primary underlying myocardial weakness as well. His course, 7½ years after the onset of failure, might be compared with that in rheumatic heart disease in many cases listed in Figure 1 that likewise first showed congestive failure, associated at the beginning with auricular fibrillation and rapid ventricular rates.

Case 2. S. G., female, aged 38. Rheumatic heart disease, mitral stenosis; paroxysmal dyspnea; acute pulmonary edema; symptoms 15 months. Course 5½ years after beginning treatment.

This patient was first seen January 4, 1933. She had apparently always been in good health and had been an exceptionally active woman throughout her life. She had traveled extensively and engaged in all kinds of activities requiring mental and physical stamina. In October, 1931, she had an attack of intense dyspnea of sudden onset while walking to a theater. This attack was accompanied by paroxysms of cough, which continued to recur for one hour and then disappeared suddenly. After this attack she noticed slight dyspnea on moderate exertion and fatigue at the end of the day. Two weeks later she had a similar attack, also precipitated by walking rapidly. Coughing was even more pronounced, and continued for almost 3 hours after the intense dyspnea had subsided. This second attack was also accompanied by severe pain in the anterior chest region and a choking sensation. During the next few days she coughed up blood-tinged sputum. Her family physician then took her to a consultant, who stressed the gravity of her condition. She reduced her activities somewhat but refused to rest in bed. Dyspnea on exertion became progressively worse, and in addition she experienced intense pain across the anterior chest region on moderate exertion. For a time this pain was so severe that she could not take ten steps. After a few months, during which she was forced by her symptoms to decrease her activity further, both dyspnea and pain became less marked. She still had dyspnea on exertion, however, and considerable fatigue at the end of the day.

At the time of examination she did not appear acutely ill. The apex of the heart was felt at the fifth interspace 10 cm. to the left. The rhythm was regular, the rate slow. The first sound at the apex was loud and booming, and in the same area there was a rough, rumbling murmur that ran throughout diastole. The blood pressure was 110/80. The lungs were clear. The vital capacity was 1800 cc.

One week later the patient complained of difficult breathing, a smothering sensation and pressure in the substernal region. Physical examination was the same except that her rhythm was totally irregular. An electrocardiogram showed fibrillation with a slow ventricular rate. She was persuaded to undergo bed rest. Approximately 24 hours after she had remained in bed she suddenly experienced intense respiratory distress accompanied by a choking sensation and slight coughing. Physical examination showed acute dyspnea, the rhythm was slow and now regular, and there were rales and sibilant rhonchi throughout both lung fields. Three injections of ¼ gr. morphine at half-hour intervals gave very little relief. Respiration continued to be labored and sibilant, and sonorous rhonchi were gradually replaced by moist rales. The lungs then gradually filled with moisture, the rales became tracheal in character, and the patient continued to bring up larger amounts of frothy blood-tinged sputum. Her condition grew steadily worse. The pulse became thready, and after 10 hours in this condition she brought up a massive pulmonary hemorrhage. With this, respiration stopped, and she lost consciousness and became cyanotic. She was immediately turned over on her side and large amounts of frothy blood were forced out of her chest by external compression. After small repeated doses of Adrenalin and caffeine, venesection and artificial respiration, breathing was re-established and she regained consciousness. The pulse gradually improved and in the next few hours the respiratory distress gradually disappeared. Moist rales in both lung fields persisted for several days. Her general condition, however, improved, and within 2 weeks she felt no discomfort save that of extreme weakness. This persisted for several weeks so that 6 weeks after the attack of pulmonary edema she had no desire to make the slightest effort. She was kept at absolute bed rest for 16 weeks and only then was given bathroom privileges. During the next 4 or 5 weeks, approximately 5 months after this attack, she was up and about at home several hours daily.

From this time on she increased her activity to the extent of going out-of-doors once daily when the weather permitted, occasionally driving a machine, and visiting friends. She did not climb any stairs or walk except when absolutely necessary, as from the sidewalk to a theater seat. She stayed in bed approximately 16 hours out of every 24.

At this time the advisability of total thyroidectomy was considered but dismissed because of a low basal metabolic rate.

Seven months after the attack of pulmonary edema she submitted to 4 weeks of absolute bed rest as a prophylaxis. In September, 1933, she went to Florida, where she led a life of restricted activity but a happy one. In the fall of 1934 she increased her activity and precipitated an attack of severe respiratory distress and wheezing that lasted several hours. She was kept in bed 12 weeks following this episode, and thereafter her activity was restricted so that she remained in bed 12 to 16 hours daily. She avoided climbing stairs, took only short walks, and spent approximately 1 out of every 6 to 8 weeks resting in bed.

It is now 5½ years since she first submitted to the prescribed treatment, and during this time except for one attack of paroxysmal dyspnea she has maintained uninterrupted good health. The rhythm has remained regular throughout this period. White-cell counts taken on two or three occasions were normal. She was kept on a maintenance dose of digitalis for 2 months after her initial attack of pulmonary edema. Thereafter digitalis was not prescribed.

Case 3. N. F., female, aged 59. Hypertension, hypertensive heart disease, cardiac asthma, congestive heart failure; marked obesity. Course 6 years after beginning treatment.

This patient was first seen March 9, 1932, complaining of progressive dyspnea on exertion, attacks of nocturnal dyspnea and edema of the legs. She had been markedly obese all her life, weighing over 200 lb. when a young girl. At times her weight was as high as 340 lb. In spite of this she was a very active, healthy woman. She raised ten children, and with the help of a younger daughter cared for a household of eleven. Six years before, she noticed some dyspnea on exertion, but this did not become progressively worse or interfere with her activities until 4 years later, when it had become more marked. Her weight had not increased; in fact, it was somewhat less. The dyspnea

gradually increased in the course of the next few months, and 4 months before she was first seen she began to have in addition attacks of severe nocturnal dyspnea. These would wake her out of sleep and force her to sit on the edge of the bed, and were accompanied by a smothering sensation. The attacks often persisted for an hour and were accompanied by a premonition of impending death. She became increasingly more orthopneic and the attacks became more frequent. Two weeks before she was seen, dyspnea had become so intense that the slightest exertion caused breathlessness, and she noticed swelling of both ankles. Physical examination showed a markedly obese woman. There were orthopnea, cyanosis and dyspnea on the slightest exertion in bed. The blood pressure was 190/100. Physical examination of the heart showed a regular rhythm, rate 100. There were no murmurs, and the heart size could not be determined by percussion. At the left lung base there were showers of moist rales. There was pitting edema of both legs. The vital capacity was 700 cc.

The patient was put on a Karell diet, digitalized and put at absolute bed rest. After the first few days of bed rest the rales at the left base and the edema disappeared; she became less orthopneic and attacks of nocturnal dyspnea did not recur. Blood pressure varied from 220/100 to 240/120. She was kept in bed for a period of 4 months. At this point lavatory privileges were permitted and her activities were slowly increased, so that 9 months after the onset of treatment she was up and about the house the greater part of the day but did not do any housework. Her weight at this time was 215 lb. Approximately 1 year after the onset of treatment she began to cook one meal a day, and except for vacations and periods of prophylactic rest she has continued to carry on in this way. It is now 6 years since the onset of rest treatment and she has not shown any evidences of circulatory failure at any time. On three occasions she has had attacks of rapid heart action persisting for periods of ½ to 2 hours. She was never seen in any of these attacks.

Digitalis was discontinued shortly after evidence of failure disappeared. It was administered for periods of a few months after each attack of rapid heart action, but otherwise she has maintained her improved condition without a maintenance dose of digitalis.

311 Commonwealth Avenue.

REFERENCES

1. Allbutt, C.: Diseases of the cardiovascular system. Musser and Kelly's *Handbook of Practical Treatment*. Philadelphia and London: W. B. Saunders Co., 1911. P. 17.
2. Allbutt, C.: Diseases of the cardiovascular system. *Ibid*. 1917. P. 476.
3. Altschule, M. D.: The pathological physiology of chronic cardiac decompensation. Medicine **17**:76-154, 1938.
4. Babcock, R. H.: *Diseases of the Heart and Arterial System*. 853 pp. New York and London: D. Appleton and Co., 1903.
5. Balfour, G. W.: *The Senile Heart; Its symptoms, sequelae, and treatment*. 300 pp. New York and London: Macmillan and Co., 1898.
6. Bamberger, H.: *Lehrbuch der Krankheiten des Herzens*. 459 pp. Wien: Wilhelm Braumüller, 1857.
7. Barringer, T. B., Jr., and Teschner, J.: The treatment of cardiac insufficiency by a new method of exercise with dumb-bells and bars. Arch. Int. Med. **16**:795-808, 1915.
8. Boas, E. P.: The treatment of early heart failure. M. Clin. North America **18**:741-758, 1934.
9. Brams, W. A.: Treatment of cardiac insufficiency. M. Clin. North America **14**:1131-1138, 1931.
10. Bramwell, B.: *Diseases of the Heart and Thoracic Aorta*. 783 pp. Edinburgh: Young J. Pentland, 1884.
11. Bramwell, C.: Treatment of congestive heart failure. Practitioner **130**:684-697, 1933.
12. Broadbent, W. H., and Broadbent, J. F. H.: *Heart Disease: With special reference to prognosis and treatment*. 331 pp. New York: William Wood and Co., 1898.
13. Brooks, H.: The failing heart: its recognition and management. M. Clin. North America **18**:727-739, 1934.
14. Brown, P. R.: The Nauheim treatment of acute and chronic heart failure. Calif. S. J. M. **5**:85-89, 1907.
15. Brunton, T. L.: *Therapeutics of the Circulation*. 274 pp. London: J. Murray, 1908.
16. Christian, H.: *The Diagnosis and Treatment of Diseases of the Heart*. Oxford Medical Publications. New York: Oxford Univ. Press, 1935. P. 160.
17. Clarke, R. M.: Cardiac care after decompensation. California & West. Med. **44**:153-157, 1936.
18. Colbeck, E. H.: *Diseases of the Heart*. 350 pp. Chicago: W. T. Keener & Co., 1905.
19. Coombs, C. F.: *Rheumatic Heart Disease*. London: John Wright & Sons, Ltd., 1924. P. 320.
20. Corvisart, J. N.: *An Essay on the Organic Diseases and Lesions of the Heart and Great Vessels*. 344 pp. Boston: Bradford & Read, 1812.
21. Davis, D., and Weiss, S.: Rheumatic heart disease; life history of severe form of disease. Am. Heart J. **10**:486-494, 1935.
22. Davis, D., and Blumgart, H. L.: Cardiac hypertrophy: its relation to coronary arteriosclerosis and congestive heart failure. Ann. Int. Med. **11**:1024-1038, 1937.
23. Dujardin-Beaumetz: *The Modern Treatment of Diseases of the Heart*. 179 pp. Detroit: George S. Davis, 1887.
24. East, C. F. T., and Bain, C. W. C.: *Recent Advances in Cardiology*. 342 pp. Philadelphia: P. Blakiston's Son and Co., 1929.
25. Eggleston, C.: The treatment of advanced heart failure. Med. Clinics N. America **4**:121, 1920.
26. *Idem*: Treatment of Cardiac Failure. Cecil's *Textbook of Medicine*. Third edition. Philadelphia: W. B. Saunders Co., 1935. Pp. 1152-1157.
27. Flint, A.: *A Treatise on the Principles and Practice of Medicine*. 1150 pp. Philadelphia: Henry C. Lea's Son & Co., 1881.
28. Fothergill, J. M.: *The Heart and Its Diseases: With their treatment*. London: H. K. Lewis, 1872. P. 205.
29. *Idem*: *The Heart and Its Diseases With Their Treatment: Including the gouty heart*. 476 pp. Philadelphia: Lindsay & Blakiston, 1879.
30. Gibson, G. A.: *Diseases of the Heart and Aorta*. 932 pp. Edinburgh and London: Young J. Pentland; New York: The Macmillan Co., 1898.
31. Greer, A. E.: Treatment of cardiac decompensation. Texas State J. Med. **17**:479-482, 1922.
32. Harrison, T. R.; Friedman, B.; Resnik, H., Jr., and Clark, G.: The effect of therapeutic measures on the cardiac output of patients with congestive heart failure. Tr. A. Am. Physicians **49**:158-162, 1934.
33. Hasebroek, K.: Zur Frage der peripherin Wirkungen der aktiven Gymnastik auf Kreislaufstörungen bei Fettherz und verwandten Zuständen; zugleich ein Versuch veränderter Auslegung der Krankengeschichte Oertels. Deutsches Arch. f. klin. Med. **94**:61-73, 1908.
34. Heimann, H. L.: Treatment of heart failure. J. M. A. South Africa **4**:418-422, 1930.
35. Hirschfelder, A. D.: *Diseases of the Heart and Aorta*. 632 pp. Philadelphia and London: J. B. Lippincott Co., 1910.
36. Hoffmann, A.: Die Behandlung der Herzinsuffizienz. Deutsche med. Wchnschr. **31**:701-705, 1905.
37. Hope, J.: *A Treatise on the Diseases of the Heart and Great Vessels*. 639 pp. London: John Churchill, 1839.
38. Hunt, G. H.: The treatment of cardiac failure. Clin. J., London **54**:205-211, 1925.
39. Jagic, N., and Flaum, E.: *Therapie der Herzkrankheiten*. 331 pp. Berlin and Vienna: Urban and Schwarzenberg, 1935.
40. Jürgensen, T.: Insufficiency (weakness) of the heart. *Diseases of the Heart*. Vol. 12. *Nothnagel's Encyclopedia of Practical Medicine*. Philadelphia: W. B. Saunders Co., 1908. Pp. 18-415.
41. Krehl, L.: Diseases of the myocardium and nervous diseases of the heart. *Diseases of the Heart*. Vol. 12. *Nothnagel's Encyclopedia of Practical Medicine*. Philadelphia: W. B. Saunders Co., 1908. Pp. 421-763.
42. Levine, S. A.: *Clinical Heart Disease*. Philadelphia and London: W. B. Saunders Co., 1936. Pp. 9, 288.
43. Lewis, T.: *Diseases of the Heart*. London and New York: The Macmillan Co., 1933. P. 26.
44. Mackenzie, J.: Rest, diet, exercise, baths. *Principles of Diagnosis and Treatment in Heart Affections*. London: Oxford Medical Publications, 1916. P. 226.
45. Neuhof, S.: *Clinical Cardiology*. New York: The Macmillan Co., 1917. P. 223.
46. Osborne, O. T.: *Disturbances of the Heart*. Second edition. Chicago: American Medical Association, 1916. P. 151.
47. Paul, C.: *Diagnosis and Treatment of Diseases of the Heart*. 335 pp. New York: William Wood and Co., 1884.
48. Paullin, J. E., and Minnich, W. R.: The treatment of congestive heart failure. South. M. J. **29**:400-403, 1936.
49. Pratt, J. H.: Rest and exercise in the treatment of heart disease. South. M. J. **13**:481-490, 1920.
50. Romberg, E. V.: *Ueber die Dekompensation der erworbenen Herzklappenfehler und ihre Behandlung*. Berlin: Julius Springer, 1929. P. 29.
51. Satterthwaite, T. E.: *Diseases of the Heart and Aorta*. 304 pp. New York: E. R. Pelton, 1905.
52. Schroetter: Changes in the position of the heart and diseases of the heart substance. *Ziemssen's Cyclopedia of the Practice of Medicine*. Vol. 6. New York: William Wood and Co., 1876. P. 175.
53. Senac, J. B.: *Traité de la Structure du Coeur, de son Action et de ses Maladies*. 2 vol. 1198 pp. Paris: J. Vincent, 1749.
54. Scherf, D.: *Klinik und Therapie der Herzkrankheiten*. 232 pp. Wien: Julius Springer, 1935.
55. Sibson, F.: cited by Pratt.[49]
56. Siebeck, R.: Die Beurteilung und Behandlung Herzkranker: Der erste Ansatz der Behandlung bei Herz-schwäche und Stauung. München. med. Wchnschr. **81**:1791-1793, 1934.
57. Starling, E. H.: *The Law of the Heart*. Linacre Lecture. 27 pp. London: Longmans, Green & Co., 1918.
58. Stokes, W.: *The Diseases of the Heart and the Aorta*. 689 pp. Philadelphia: Lindsay & Blakiston, 1854.
59. Strauss, A. E.: Treatment of myocardial insufficiency. M. Clin. North America **20**:547-558, 1936.
60. Warfield, L. M.: The treatment of the patient who has heart failure. Wisconsin M. J. **34**:546-552, 1935.
61. Wearn, J. T.: Tr. Assn. Am. Phys. (in press).
62. Wilson, W. J.: Treatment of cardiac decompensation. Detroit M. J. **19**:14-17, 1918.
63. Wynn, J.: Treatment of cardiac decompensation. J. Indiana M. A. **18**:311-317, 1925.
64. Daly, P. A., and Strouse, S.: The so-called medical complications of pregnancy. J. A. M. A. **96**:1655-1659, 1931.
65. White, P. D.: *Heart Disease*. 744 pp. New York: The Macmillan Co., 1937.

TWO YEARS OF DIABETIC SURGERY

BERNARD RABINOVITZ, M.D.,* AND JACOB WEISMAN, M.D.†

SPRINGFIELD, MASSACHUSETTS

SINCE the advent of insulin therapy the life of the diabetic has been considerably prolonged, and he is thus rendered more liable to the accidents of life which require surgery. Increased longevity has also made it possible for the diabetic to develop arteriosclerosis and its sequelae: gangrene, ulcers and infection. Previous to the insulin era the diabetic was denied all but the most peremptory surgery. Now the benefits of surgery are granted to all diabetics, and even urged upon them, since in some cases elimination of infectious foci and of the causes of disordered metabolism tend to ameliorate their diabetic condition. Thus cholecystectomies and thyroidectomies are performed with very much less danger than previously. Statistics show that 50 per cent of all diabetics require surgical treatment at one time or another.[1]

Reviewing the literature[2] from 1895 to 1926, we find a total of 1864 operations on diabetics with a mean mortality of 25.6 per cent; this percentage ranged from 9 to 40, with most of the reports in the higher brackets. During Naunyn's era the mortality, up to 1912, was as high as 80 per cent. Hence arose the popular belief that surgery of the diabetic was peculiarly hazardous. Joslin[1] in 1928 stated that "the surgical diabetic is the diabetic who dies."

Many factors have heretofore contributed to the death of surgical diabetics. These factors, usually physiological, can in great part be combated by modern methods of treatment. Frequent causes of death were dehydration, acidosis, coma, anuria, circulatory collapse, septicemia, hepatic insufficiency and cardiovascular incompetence. Since 75 per cent of surgical diabetics are over forty years of age, they are poorer risks for surgery which comes late in life. Also, since diabetics develop arteriosclerosis at least ten years in advance of non-diabetics,[1] we are dealing with people three-fourths of whom are physiologically beyond the fifth decade. Previous to the discovery of insulin, diabetics were starved and poorly nourished, resistance to intercurrent infection thus being impaired. Overhospitalization and excessive restraint in bed resulted in a lowered morale and decreased vitality; it increased the need for insulin and retarded healing. These factors are mentioned so as to direct the reader's attention toward the disordered physiologic factors involved.

*Metabolist, Springfield Hospital.
†Chief of Welfare Clinics, City of Springfield.

PHYSIOLOGY

One of the foremost complicating factors of diabetics is hepatic insufficiency. The medical literature does not sufficiently emphasize the tremendous importance of the liver in the diabetic state. First, there is a decrease in the deposit of glycogen upon which the organism must draw for its energy.[6] There is usually hepatomegaly due to fatty infiltration, which is antagonistic to the storage of glycogen.[3] Consequently there is no desaturation and dehydrogenation of fatty acids, the sequelae being an increase in circulating acetone, diacetic and β-oxybutyric acids, which results in acidosis. Cholesterol is not broken down, and the result is an abnormal deposit of this compound in the vascular system, and cholelithiasis. Warren[4] demonstrated the effects of increased cholesterol deposits in the gall bladder. Of 245 diabetic autopsies, 62 cases revealed cholelithiasis; in 400 nondiabetic autopsies the incidence was only 54 cases. The gall-bladder disease in itself may then further inhibit the function of the pancreas, thus aggravating the diabetic state. Only a portion of the lactic acid is converted into glucose and glycogen, so that acidosis is increased; deaminization of amino acids is incomplete, and the formation of urea is decreased. The abnormal liver fails to destroy toxins, with a resultant increase in toxemia. There may be decreased production of the insulinogenic kinase which activates insulinogen, the precursor of insulin, making the diabetic more insulin insensitive.[5] Such kinase has not been definitely proved to exist.

All the foregoing disordered physiologic factors contribute to the dangers of diabetic surgery.

When kidney function is good the diabetic rarely develops coma and acidosis. The damage to the kidney which results from irritation by polyuria due to sugar, ketone bodies and other uncombined inorganic acids is the cause not only of uremia and anuria but also of coma. With such irritation there is cloudy swelling of the renal tubules, albumin, "coma casts,"[1] decreased urea output, decreased ammonia formation and depletion of sodium, potassium, calcium and magnesium stores. With still further damage the glomeruli cease functioning and there is little or no excretion of ketone bodies and other waste materials. Albumin and casts disappear, and anuria and uremia ensue.

It is well known that in diabetes the heart is

frequently damaged by sclerosis of the coronary arteries. Warren[4] cites 270 cases 124 of which showed coronary sclerosis; Wilder[7] reports 17 out of 49 cases; Nathanson[8] reports coronary sclerosis in 52 per cent of his diabetics. In the sclerotic heart the myocardium is weakened and its conductivity may be reduced; there is also a relative anoxemia of the heart muscle as a result of the narrowed and sclerotic condition of the coronary vessels, the diabetic thus being made prone to angina pectoris.

The lungs in diabetic surgery are also vulnerable. Hypostatic and congestive phenomena are common when the patients are kept in bed for too long a time, since most of them are beyond middle age.

The defense mechanism is also put out of gear; the low resistance to infection and decreased vitality of the diabetic are common knowledge. There is less phagocytosis and less defense power;[3] the presence of infection decreases insulin utilization and increases hormones antagonistic to insulin; pus partly neutralizes the insulin effect, decreases glycogenesis, and increases glycogenolysis. Infection may also destroy the hypothetical insulin kinase mentioned previously.

To Joslin we owe the division of diabetic surgery into two types, the preventable and the nonpreventable. Under "preventable" we include all surgical diseases resulting directly from the diabetes and its associated metabolic disorders, for example infection, cataracts, gangrene. Under "nonpreventable" we include those which cannot be prevented by proper diabetic management. In this class we have appendicitis, thyroid disorders, pelvic operations. Still another classification may be applied: surgery may be emergency as in ruptured gastric ulcer, or elective as in perineorrhaphy, or prophylactic as in cholecystectomy.

PREPARATION OF THE PATIENT

Preoperative preparation must be entirely individualized to suit the patient and the type of diabetic surgery to be performed. In cases of elective surgery the patient is not kept in bed, but is allowed to walk about in order to improve his morale and general vitality. His tolerance is studied by noting his urinary response to his required diet. The necessary amount of insulin is given to keep him sugar-free; if he appears to be dehydrated, he is allowed more sodium chloride and fluids. The diet usually consists of from 200 to 300 gm. of carbohydrate, with low-fat content and protein according to the requirements of the individual and the condition of his kidneys. The afternoon preceding the day of operation, usually about one hour after lunch, the blood-sugar content is determined. If infection is present it is wise to resort to surgery as soon as possible so that the infectious foci may be eradicated and the general diabetic condition improved. In these infected cases, especially when anemia is present, a preliminary blood transfusion is considered a worthwhile prophylactic measure. When sedation is necessary, we prescribe chloral hydrate. Barbiturates are avoided whenever possible. (If urine is tested by Fehling's solution, we must remember that chloral hydrate may give a false positive test for sugar.)

In the emergency case, where time is pressing, a urine specimen is at once examined for sugar; catheterization should be done if necessary. A sample of blood is also taken, and the blood sugar should be determined immediately. In the extreme case a blood transfusion may be given first, as mentioned above, but usually a hypodermoclysis will suffice; glucose, if given, must be covered by an adequate amount of insulin. Frequently twice the required amount of insulin is given in order to combat the acidosis resulting from anesthesia.

If spinal or regional anesthesia is to be used, we follow this scheme: two hours before operation, the usual dose of protamine-zinc insulin is given; one half hour later regular insulin is given in amounts sufficient to cover the 40 or 50 gm. of soluble carbohydrate given one hour before operation (orange juice and glucose).

If general anesthesia is to be used, protamine-zinc insulin is given three and a half hours before operation, regular insulin three hours before and carbohydrate two and a half hours before, so that the stomach will be empty when the anesthesia is administered. Hypodermoclysis is given where food by mouth is inadvisable or rejected. Before leaving for the operating room all patients are catheterized, if unable to void, and the urine is examined for sugar and acetone. Where acidosis is suspected but not evident from the urine examination because of excessive kidney damage, it has been deemed prudent to examine the blood serum for acetone and indican.

CHOICE OF ANESTHETIC

The choice of an anesthetic is important because of its effect on the water balance and pH of the blood. All methods of general anesthesia may disturb the metabolism of the diabetic. As McKittrick and Root[2] have pointed out, the acid-base equilibrium may be influenced by variations in three bodily functions; respiration, renal excretion and carbohydrate metabolism. With deepening of respiration, acidosis results both from the blowing off of carbon dioxide and from the anesthesia itself. With renal irritation and hypofunc-

tion, there is a loss of alkali reserve due to diminished production of ammonia. Even in the normal person alterations in carbohydrate and fat metabolism are common after ether anesthesia. Thus there is an increase in blood sugar and in ketone bodies.

Whenever possible we have tried to use regional or spinal anesthesia. These are the safest for the surgical diabetic. Local anesthesia, if used, should not infiltrate diseased tissue. Where general anesthesia is necessary, one should make certain that the patient has been well prepared with fluids and glucose. During anesthesia, sufficient oxygen must be supplied to avoid anoxemia. Loss of fluid by perspiration and respiration, if excessive, should be replaced by hypodermoclysis. Occasionally 10 or 15 units of insulin may be given during the anesthesia. The choice of anesthetic must also depend upon the cardiocirculatory competency of the patient.

OPERATION

With arteriosclerosis and lowered resistance to infection, the diabetic must frequently submit to amputation of an extremity or one of its parts. By x-ray visualization of peripheral vascular sclerosis, palpation for surface temperature, histamine test and oscillometry, we may determine whether surgical or medical treatment holds more promise; also the level at which amputation should be performed if and when deemed necessary. In gangrene, if the circulation appears to be fairly good and there is no evidence of bone destruction, we have thought it prudent to give medical treatment a fair trial. If amputation must be performed, the patient is kept in bed; bedclothes are kept away from the tissues by a cradle, but without heat, moist or dry, and without elevation, and with the heel protected against pressure. Wet dressings are forbidden, as they may cause maceration. At times we have used artificial gastric-juice* dressings for lysis of necrotic tissue. For exercising the body and increasing bodily vigor, a Balkan frame with trapeze is attached to the bedstead. Operations on the lower limbs are usually performed under spinal anesthesia, no tourniquet being applied and ligation of blood vessels being kept to a minimum. Smith[9] reports a mortality of 18 per cent in his series of leg amputations. In our 10 cases, in the majority of which mid-thigh amputations were done, the mortality was 10 per cent.

Our patients are urged to sit in a wheel chair on the third day and to get up on crutches as soon as the sutures are removed. Prophylaxis, so far as the remaining limbs are concerned, is impressed upon the patient, who should perform his Buerger's exercises with great fidelity thereafter. The need of instructions regarding care of the feet for all diabetic patients at the hospital cannot be overemphasized.

In abdominal operations, for example appendectomy and hysterectomy, anesthesia should be as light as possible, and surgery as dexterous as possible. Exposed tissues should be kept warm and moist; all tissues must be handled gently. In cases of cholecystitis the patient should be kept on a very high carbohydrate, fat-free diet for three or four days, and should be allowed out of bed if possible. With a long and shock-producing operation the patient should be given a hypodermoclysis immediately afterward. Where an emergency appendectomy must be performed, a blood transfusion before operation is extremely valuable and the use of local or regional anesthesia, when possible, is most desirable. In thyroidectomy for hyperthyroidism the difficulties are manifold, as the thyroid toxicity has served to decrease glucose tolerance, and removal of this gland may cause a rapid reversal of metabolic conditions, resulting in hypoglycemic shock. The surgical removal of enlarged prostates presents a difficult problem in that here we are dealing with aged men, usually debilitated, arteriosclerotic and hypertensive, whose kidney excretion is poor and whose renal threshold is high. The latter deficiency may result in sugar-free and acetone-free urine, making the tests for acetone and indican in the blood serum almost imperative. The prostate patient should be allowed to walk about as soon as practicable after operation. Spinal anesthesia is, of course, the method of choice.

POSTOPERATIVE CARE

After operation, even more than before, the treatment must be wholly individualized. Fifteen hundred cubic centimeters of 5 per cent glucose in saline is administered subpectorally shortly after the operation — rapidly at first, then more slowly. If the case is an abdominal one, intravenous drip may be advisable, the glucose to be covered by a sufficient amount of insulin. If able to take food by mouth, the patient is given concentrated soluble carbohydrate, liquids and sodium chloride every two hours; for example ginger ale, orange juice and glucose, skimmed milk and gruels, also bouillon if dehydrated. The urine should be examined every three or four hours during the next twenty-four hours. At times the blood-sugar content is determined about the middle of the afternoon of the first day. If anuria results from the anesthesia one may have to resort to the use of hypertonic saline (10 per cent). We have often attempted to administer 200 gm. of carbohydrate

*Dilute hydrochloric acid 18 gm., pepsin 20 gm. and glycerin 5 gm., in 1000 cc. of water.

within twenty-four hours postoperatively. At this time a nurse with excellent training is important — she must watch constantly for signs of acidosis and hypoglycemic shock. The intern also must be trained to recognize aberrations from a smooth course. He should watch for increased tolerance after the removal of an infectious focus and be prepared to combat the incidental hypoglycemia.

A highly important observation has been made in our series of cases. If after the removal of an infected area, for example a carbuncle, there should be no subsequent increase in carbohydrate tolerance, as manifested by a need for less insulin, a pus pocket, improper drainage or new areas of infection should be sought. In order to prevent decubitus, rubber bed sheets are forbidden. Deep breathing is encouraged to prevent atelectasis and frequent change of position is urged. On the second day there is a consultation between internist, dietitian and patient. They decide upon the most agreeable type of soft-solid diet, having in mind the state of the patient's dentition and his vagaries as to appetite. It is not sufficient to order any diet, for example C200, P50, F40 — there must be the utmost co-operation between the diet kitchen and the internist. So also must there be a mutual working partnership between the internist and the surgeon.

As soon as it is safe, the patient is allowed to sit up in bed and to take some exercise on the Balkan frame and trapeze. The meals are then gradually reduced from six to three a day, with insulin or protamine-zinc insulin to control the glycosuria. The patient is allowed out of bed as soon as possible, in order to increase his morale and sense of well-being. He is then carefully instructed as to diet, insulin, care of feet and other prophylactic measures before he is discharged to the diabetic clinic.

SURVEY OF CASES

During 1936 and 1937 at the Springfield Hospital, we had a total of 80 operations on diabetics; tonsillectomies, dental extractions and other minor procedures have been omitted from this review. The 80 operations were performed on 76 individuals, of whom 52 were females and 24 males — a ratio of about 2:1. The age distribution is seen in Chart 1 and supports the statement that 75 per cent of surgical diabetics are over forty years of age. Forty-five operations were classed as non-preventable, 35 as preventable (Table 1). The types of operation performed varied from hemorrhoidectomy to subtotal gastrectomy. Thirty-eight operations were performed under general inhalation anesthesia; 23 under spinal and regional anesthesia; and 19 under local anesthesia.

In the group of the 45 non-preventable operative cases there were 3 deaths — 2 following appendectomy and 1 after a nephrolithotomy. The 35 preventable operations were followed by 1 death — a case of mid-thigh amputation. The 4 deaths were all in private cases, agreeing with the report of Standard, Brandaleone and Ralli,[10] whose mortality figures for clinic and private cases are 6.9

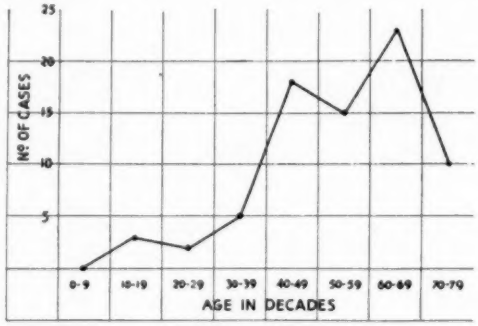

Chart 1.

and 20.8 per cent respectively. The mortality of our small series was 5 per cent. Joslin,[11] in a series of 1030 cases, reported a mortality of only 6.4 per cent, a remarkably low figure for such a large number of cases.

Table 1. *Types of Operation in Diabetic Surgery. 1936-1937.*

NON-PREVENTABLE OPERATIONS	NO. OF CASES	NO. OF DEATHS	PREVENTABLE OPERATIONS	NO. OF CASES	NO. OF DEATHS
Prostatic resection	5		Incision of abscess	5	
Orchidectomy	3		Incision of carbuncle	6	
Ventral hernia	1		Incision of infected finger or toe	5	
Eye operation	1		Amputation of leg	10	1
D and C; pelvic repair	6		Amputation of finger	2	
Appendectomy	6	2	Amputation of toe	6	
Herniorrhaphy	3		Cataract	1	
Therapeutic abortion	1				
Amputation of penis	1				
Excision of cartilage	1				
Amputation of breast	1				
Pelvic operation	6				
Nailing hip	1				
Skin graft	1				
Gastrectomy	1				
Nephrolithotomy	2	1			
Hemorrhoidectomy	1				
Exploratory laparotomy	1				
Mastoidectomy	1				
Cholecystectomy	2				
Totals	45	3		35	1

Following are the brief summaries of our fatal cases.

Case 91,942. A 65-year-old diabetic woman was admitted to the hospital March 7, 1936, with gangrene of the toe. She refused mid-thigh amputation. The toe was amputated under spinal anesthesia. On the 9th postoperative day there was infection and extension of gangrene. Mid-thigh amputation was then performed, but the patient died of septicemia 24 hours later.

This patient was unco-operative in that she did not follow her diet, left off insulin against advice, refused hospitalization when gangrene developed, and did not permit mid-thigh amputation when advised. The death was probably avoidable.

Case 125,420. A 60-year-old woman was admitted to the hospital August 22, 1936, with renal symptoms; she was not a known diabetic, and the urine was negative for sugar on several examinations. Pyelography revealed a renal stone and a dilated ureter on the right side. Nephrolithotomy was performed under ether anesthesia. The postoperative condition was poor. The patient was drowsy for several days, and study revealed diabetic acidosis. In spite of heroic treatment, the patient developed acute thyroiditis and died on the 9th postoperative day.

This case demonstrates the fact that with renal insufficiency the urine may be sugar-free and acetone-free in the face of an existing diabetes. Had the diabetic condition been known before operation, death might have been prevented.

Case 125,799. A 64-year-old man was admitted to the hospital September 20, 1936, with acute gangrenous appendicitis. The presence of diabetes had been known for 1½ years. The preoperative preparation was of necessity hurried and incomplete, as the patient's condition was grave. Operation, under ether anesthesia, consisted of drainage of the abdomen without removal of the appendix. Death occurred 72 hours postoperatively.

Here we dealt with a poor risk and a grave situation, even discounting the diabetic state. The death may be classed as unavoidable, although the preparation was not quite what we believe to be complete.

Case 130,835. A 66-year-old woman was admitted to the hospital September 12, 1936, with an acute condition of the abdomen. Diabetes had been recognized for 2 years. Operation was performed with very little preparation, and a gangrenous appendix was removed under ether and local anesthesia. The diabetic management remained in the hands of a non-staff doctor until the patient's condition became very poor. Death occurred 36 hours postoperatively.

Apparently the necessity of an immediate operation overshadowed the usual advisability of preoperative preparation. This omission was imprudent, and we must consider this death as probably avoidable.

The following case is reported because it well demonstrates the extent of major surgery which can be successfully performed on the diabetic patient.

Case 126,284. A 66-year-old woman, weighing 209 lb., was admitted to the hospital October 26, 1936, with a history of hematemesis. Diabetes had been recognized for 15 years. The blood-sugar content after a test meal was 286 mg. per cent. The patient was placed on a high-carbohydrate, low-fat, ulcer diet and insulin. It took 8 days to render the patient's urine sugar-free and to lower the blood-sugar content. X-ray studies revealed a large, filling defect on the lesser curvature of the stomach. The diagnosis was neoplasm without obstruction. The patient was typed for possible transfusion and proper donors were held in readiness.

On the day of operation the patient received 40 units of regular insulin at 5:30 a. m., 300 cc. of orange juice at 6 a. m., and 10 gm. of glucose by mouth at 9 a. m. Dr. Frederick Sweet performed the operation under nitrous-oxide, oxygen and ether anesthesia, employing the Connell re-breathing machine. The operation was the Polya type of subtotal gastrectomy. Immediately after operation saline and glucose with adequate insulin were administered subpectorally. Next, a continuous intravenous drip was started and insulin was given every 2 hours; Wangensteen nasal suction also was instituted. A total of 27,500 cc. of 5 per cent glucose in saline solution was given in 5 days. On two occasions, because of difficulty in getting into a vein, glucose was discontinued, but insulin was continued. There were two severe episodes of hypoglycemic shock. These were successfully treated with 50 per cent glucose given intravenously. The blood-sugar contents on these occasions were 66 mg. and 20 mg. respectively. On the 5th postoperative day nasal suction and intravenous drip were discontinued, and small oral feedings of soluble carbohydrate and protein were begun. Convalescence was rapid, and the patient was discharged improved on the 28th postoperative day.

SUMMARY

In diabetic surgery the patient is an individual whose physiologic functions and metabolism are in an extreme state of imbalance. The important role played by the liver has been emphasized; we have also discussed some of the other disordered physiologic functions which contribute to the hazards of this type of surgery.

We agree with Abramson[12] that diabetics cannot be treated in accordance with general principles only and emphatically urge individualization of treatment. Preoperative preparation with attention to detail, expert anesthesia, adept surgery and careful postoperative management are factors which will make diabetic surgery as safe as non-diabetic.

This report deals with 80 cases operated on in 1936 and 1937 with a mortality of 5 per cent. Protocols of fatalities are presented in brief. We stress the necessity of co-operation between the internist and the surgeon if a low-mortality percentage is to be attained. We wish to emphasize also that surgery of the diabetic should be done only in hospitals properly equipped to perform prompt and accurate laboratory tests, since we are dealing with surgery that is always of an emergency character.

We are indebted to those surgeons of the Springfield Hospital whose cases we were permitted to review.

REFERENCES

1. Joslin, E. P.: *Treatment of Diabetes Mellitus.* Fourth edition. 998 pp. Philadelphia: Lea & Febiger, 1928.
2. McKittrick, L., and Root, H.: *Diabetic Surgery.* Philadelphia: Lea & Febiger, 1931.
3. Grafe, E.: *Metabolic Diseases and Their Treatment.* 551 pp. Philadelphia: Lea & Febiger, 1933.
4. Warren, S.: *The Pathology of Diabetes Mellitus.* 212 pp. Philadelphia: Lea & Febiger, 1930.
5. Himsworth, H. P.: Activation of insulin. Lancet **2**:935-936, 1932.
6. Sherrill, J. G.: The diabetic as a surgical risk. Anesth. & Analg. **7**:382-385, 1928.
7. Wilder, R. M.: Necropsy findings in diabetes. South. M. J. **9**:241-248, 1926.
8. Nathanson, M. H.: Coronary disease in 100 autopsied diabetics. Am. J. M. Sc. **183**:495-502, 1932.
9. Smith, B. C.: Therapy of surgical complications of diabetes mellitus at Presbyterian Hospital in New York City, 1930-1935. Surgery **2**:509-518, 1937.
10. Standard, S.; Brandaleone, H., and Ralli, E. P.: Surgical results in treated and untreated diabetic patient: And analysis of altered prognosis in a comparative group of 474 clinic and nonclinic patients. J. A. M. A. **110**:627-629, 1938.
11. Joslin, E. P.: personal communication.
12. Abramson, P. D.: Insulin and surgery. Ann. Surg. **96**:49-69, 1932.

RAGWEED-POLLEN SURVEY IN MAINE FOR 1937

CHARLES B. SYLVESTER, M.D.,* AND O. C. DURHAM†

PORTLAND, MAINE, AND NORTH CHICAGO, ILLINOIS

OUR chief motive in making a survey of atmospheric pollen in Maine was to promote a campaign for intelligent eradication of the weeds, but the data will doubtless prove of value to physicians engaged in preventive treatment and to victims of ragweed hay fever who wish to find a favorable refuge. We hope that this report will be the means of a wider appreciation of the buoyancy of ragweed pollen, which often rides at heights of several thousand feet and is carried for hundreds of miles. In Maine, as well as in other states, there have been spasmodic efforts by towns, counties and districts to destroy ragweed. These righteous endeavors would have been more effective if the magnitude of the problem had been better understood and if there had been co-ordination of effort on a larger scale.

The geography of Maine must be noted in order to see that the state is remarkably isolated by seashore and forested mountains where no ragweed can originate. The boundaries of the state are largely protective, and it is apparent that no state in the ragweed area east of the Rocky Mountains and north of Florida could so easily maintain a quarantine against ragweed pollen.

Not only does Maine have its very long ocean shore to the south and east, but it has to the west the White Moutains, continued in a long mountainous range across the northwest of the state. The extensive forest areas of Maine are also situated in the west, northwest, north and in the eastern margin of the state. The vulnerable border is evidently the southwest, between the coast and the White Mountains. On the northeast, New Brunswick is adjacent, sparsely settled, with few lines of communication, and usually with westerly winds. Also it may be that there is more frugal farming, with fewer neglected, uncultivated fields. Whatever reason is assigned, our survey evidences little dissemination of pollen across the Canadian border.

It is interesting to note that ragweed appears to have been introduced along the more frequented lines of travel. A theory has been advanced that railway cars and automobile tops with their ridged edges were carriers of pollen from distant states. The more likely explanation is that the increasing number of small farms, gardens and roadside ditches furnish ideal culture beds for ragweed and are thus a local source of air contamination. These, with the pollen brought in by prevailing westerly winds, account for our finding of a relatively high incidence of pollen in the southwestern part of the state.

In his nation-wide atmospheric pollen studies one of us (O. C. D.[1]) exposed pollen slides during 1934 and 1935 at Bar Harbor and at Upper Dam on the Rangeley Lakes, also in 1935 at East-

Figure 1. *Map of Maine, showing the mountainous boundaries and heavily forested areas.*

port. One of us (C. B. S.) exposed slides in Portland during August and September, 1936, but we afterward decided that the shelter which had been used was not suitable, so a plan was arranged for a co-operative study in 1937. Dr. George H. Coombs, director of the Maine Bureau of Health, was immediately concerned in the success of such an air survey, and wished to make it state-wide in extent. The bureau furnished the expense of necessary material, and the labor in placing and collecting slides. The selection of receiving stations was made with the purpose of securing data from differing representative areas, such as

*Consultant in allergy, Maine General Hospital, Portland.
†Chief botanist, Abbott Laboratories, North Chicago.

the coast with its islands and headland resorts, the central and northern farming section, the cities and the extensive forests. However, it was obvious that we should make tests only where people are present in reasonable numbers in August and September.

Ragweed counts having been published for Rangeley Lakes, Eastport and Bar Harbor, these important points were not again included. The regions first checked on the map of Maine were Aroostook County, Moosehead Lake, the deeply indented coast to the east of the Kennebec outlet, the Penobscot Bay shores and the more heavily

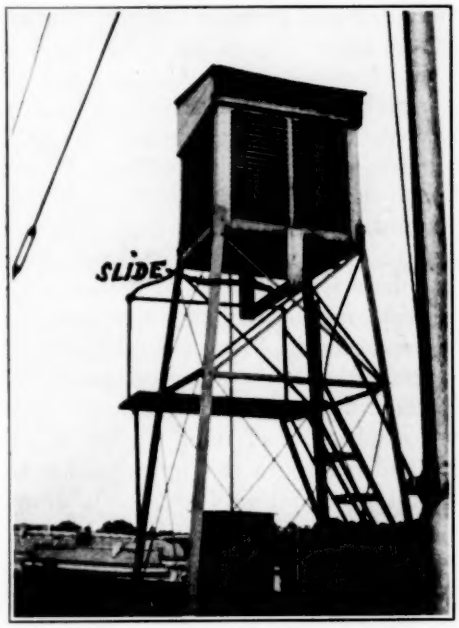

Figure 2. *United States Weather Bureau instrument shelter at Portland. The block holding the slide rests on the bar under the shelter.*

populated southwestern corner of the state. The stations finally placed were those at Presque Isle, Greenville, Poland Spring, Augusta, Machias, Camden, Southport, Portland and York. Camden is omitted from our tabulations as it did not qualify for the entire season; its exposed slides showed a low count consistent with that of the other shore towns.

From previous clinical observations we expected the season to begin August 10. However, to check up our accuracy, we made our exposure August 8, and daily thereafter for fifty days. Results showed August 10 to be early enough for the first exposure. Uniform directions were given with each box of slides for the proper handling without contamination. The slides were coated with a thin film of white vaseline and labeled with the station and consecutive dates. They were exposed in a horizontal position, held in an ordinary clothespin clip fastened to a base. Protection from rains was afforded by a roof only. Slides were changed each morning. We took the precaution, as a proof of technic and identity, to have each pollen-receiver photographed, close up and distant, showing the elevation and freedom from lateral contamination and air deflection. The custody and care of the slides were placed in the hands of authorized observers at weather bureaus, fire stations, a state sanatorium, a state normal school, a C.C.C. camp, the state capitol and two resort hotels.

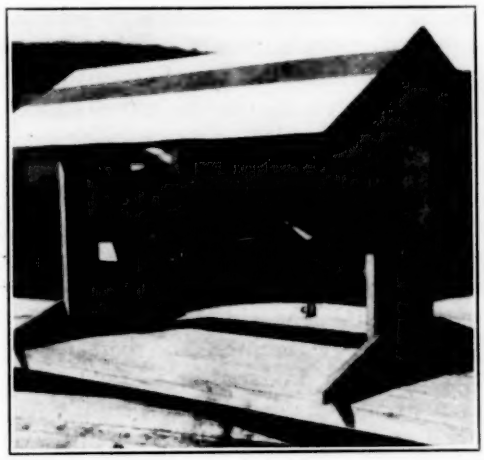

Figure 3. *Shelter used for the pollen slides exposed on the State House at Augusta.*

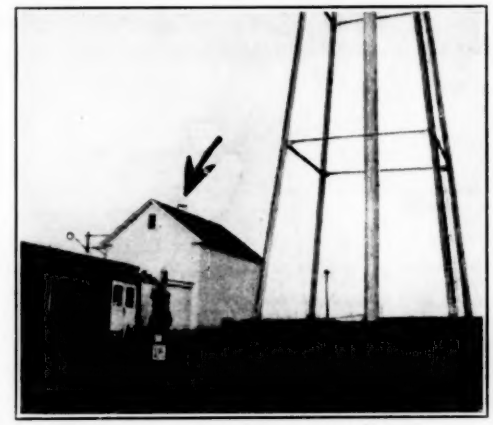

Figure 4. *Pollen survey station at Northern Maine Sanatorium, Presque Isle, Aroostook County. Note shelter on top of building.*

Counting was done by a standard method used by Durham[2] in his nation-wide studies: contiguous microscope fields in five separated paths across the width of the slide, approximately an area of 1.8 sq. cm. All slides were counted by our technicians in Portland and rechecked by one of us (O. C. D.).

Contamination of the air with pollen depends on two main factors: pollen production and pollen distribution. For abundant production there must be a reasonably large acreage of either waste land or farm land devoted to cereals. The character of the soil, the amount of rainfall and the average temperature also have much to do with the maturation of the pollen crop. Distribution of the matured pollen depends mostly on wind currents. Convection currents are mostly responsible for lifting the pollens to the higher altitudes and lateral currents for long-distance transportation. Thus while a given locality may be entirely free from local growths of ragweed because of unfavorable soil, or occupancy of the land by grass, forests or buildings, the air may be periodically or continuously contaminated by pollen brought in by the wind. The air of a large city is polluted, even in the business and best residential sections, by pollen produced in vacant lots, in the subdivisions and on adjacent farm lands. An undisturbed forested area within fifty miles of an agricultural area will experience periodic showers of ragweed pollen during the ragweed season when strong winds blow directly from the agricultural to the forested area. The small town or city which does not have subdivisions gets its pollen from surrounding agricultural lands or in some cases from distant weedy areas.

Much of the essential data obtained in the 1937 survey will be found in Table 1, in which are included for purposes of comparison average figures for New York City and Boston, as well as counts made in 1934 and 1935 at Bar Harbor, Eastport and Upper Dam. Hay-fever symptoms may be experienced when the pollen count is less than 25, but 25 has been chosen as a minimum figure for counting the number of hay-fever days in a given locality. For purposes of comparison any reasonable figure would do. The total count is obtained by adding the figures for each day through the season. The "ragweed index" is figured on the same basis as was used in a previous nation-wide evaluation of hay-fever-resort areas. On this arbitrary scale, some mid-western cities have index figures of 100 or more. Examples follow: Buffalo 100, Indianapolis 124, Toledo 96, Chicago 74. The figure is obtained by assigning one point for each day when the count reaches or exceeds 25, one point for each 100 grains of pollen on the day of the highest count and one point for each 200 grains of pollen in the seasonal total.

The focus of local atmospheric ragweed-pollen contamination in Maine would seem to be in the agricultural area of the southwestern part of the state. Portland is really the only point studied where the ragweed problem approaches in severity that of the large cities of the central and eastern states. In Portland a considerable part of the pollen is probably from vacant lots and untenanted subdivisions, although ragweeds will be found along public roads and in gardens at some distances into the more rugged portions of the state.

An annual total fall of less than 500 ragweed-pollen grains may be considered a very good record, but there is no guaranty that favorable conditions will continue without active measures for control of ragweed. Many localities, which were once entirely free from atmospheric contamination, have become badly contaminated in the course of a few years because of utter neglect of adjacent gardens and fields. In the agricultural section of the central states ragweed control is a practical impossibility, but in a district where the acreage of tilled land is small, or the land kept free of weeds, co-operative endeavor could result in decided reduction of the pollen content of the air.

A noticeable feature of the behavior of the 1937 ragweed season in Maine is the three periods of atmospheric contamination (Fig. 5). The first occurred about August 20, with almost a maximum count in Portland, York and Southport. The second and heaviest period of high concentration occurred during the first week of September, with

Table 1. *Ragweed Pollen Counts in Different Localities.*

CITY	POPULATION	NUMBER OF HAY-FEVER DAYS	HIGHEST COUNT FOR ONE DAY	TOTAL RAGWEED COUNT	RAGWEED INDEX
Portland	70,810	18	114	1,225	25
York	2,532	5	78	348	8
Southport	412	5	65	369	8
Poland Spring	580	11	75	788	16
Augusta	17,198	6	57	573	9
Machias	1,853	2	39	250	4
Greenville	1,614	3	46	223	4
Presque Isle	6,965	0	14	84	1
Bar Harbor	4,486	5	76	426	8
Eastport	3,466	4	43	280	6
Upper Dam (Rangeley Lakes)	78	1	69	139	2
New York City	6,930,000	27	373	1,675	39
Boston	781,000	10	140	790	15

maximum counts at all stations. A minor flare-up occurring on September 15 was particularly evident at Portland, Augusta and Poland Spring, but was felt at all stations. It seems quite evident that these three showers were state-wide. The wind velocity at the various stations at the time of these three showers was not particularly high, but a careful study of the records shows that the prevailing wind direction throughout the state on August 20 was west, and on September 2 and 15 south or southwest. It would seem, then, that the pollen showers can be accounted for on the basis of wind direction rather than wind velocity.

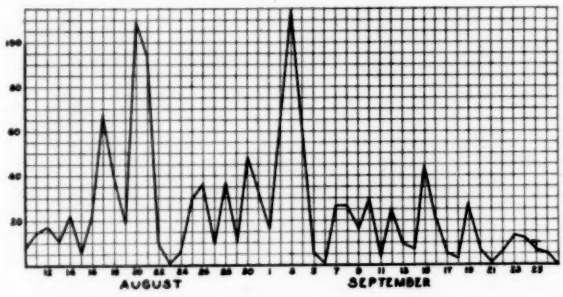

Figure 5. *Ragweed pollen season of 1937 at Portland. The three periods of state-wide atmospheric contamination are clearly shown in this record. The scale refers to the number of ragweed pollen grains found on an area approximately 1.8 sq. cm. on each slide.*

The time of maximum pollen production in Maine coincides very well with that in adjacent eastern states. The climax on September 2 coincides with the date of highest ragweed pollen incidence in 1937 in New York City. As an example of the abundant evidence in this study of pollen transport, we may cite the following facts: The slides were exposed in Poland Spring on a tower 120 ft. high. There is little local growth of ragweed, yet on September 2, 3 and 4 and again on September 15, the count at Poland Spring varied from 54 to 75. Between these two waves the concentration at Poland Spring dropped to 5 and 6 and in one instance to 0.

The surprise of our survey lies in this: the cities have more atmospheric ragweed pollen than has the country. Within a dozen miles of Maine's largest city can be seen acres of neglected fields with a bountiful crop of ragweed and little else, while in the rich agricultural Aroostook County, tillage land is too valuable to neglect. Our survey shows that Aroostook County has the lowest ragweed count in the state. Most soils, if not made to produce crops, will produce ragweed. It is evident that soils, like human beings, should be kept busy.

There is an astonishing ignorance, even in the medical profession, of the identity of the growing ragweed. It is so common and homely and generally dust-covered that it is unrecognized. Attention should be called to the under surface of the blossoms of the maturing plant, with its thousands of tiny yellow anthers bursting with pollen. The enormous pollen-producing power of the ragweed should be understood. Photographs have been published of the ragweed-lined sidewalks of Cleveland, Ohio, which has an atmospheric load measured in tons of pollen. The back yard and the roadside and edges of sidewalks are commonly beds of ragweed. The public must learn to appreciate the unseen atmospheric load of pollen and the ubiquitous ragweed. The seeds remain viable for many years, ready to spring up in every break in the soil. Vacant lots, back yards, gardens, roadsides and neglected fields offer a thousand opportunities.

Maine places "Vacationland" on its automobile plates, and the state is filled with summer camps for boys and girls. It should protect its reputation as a health resort. It has every natural advantage. Many seek freedom from hay fever, and many claim to find it at mountain and shore resorts. On strict analysis, this freedom is found to be actually relative, with some clinical manifestation, even if so temporary or slight as to be happily forgotten. The public should learn that not "climate" but pollen pollution of the air causes hay fever. We cannot repeat too often that ragweed has only a nuisance value. It is always a pest, can be nothing but a pest, and is in this state the sole cause of late summer and fall hay fever.

The survey and report of a public-health nuisance should be followed by rational suggestions for its eradication. Ragweed is an annual, and therefore its roots do not require removal. Along the roadsides and in fields it can easily be cut, and must be burned or otherwise destroyed to prevent maturing of the pollen. Local destruction of ragweed, however thorough, must have adjacent co-operation, which in Maine should extend to the whole state.

We have shown the necessity for some knowledge of geography to explain the prevalence and distribution of ragweed, some knowledge of agriculture and soils, some knowledge of botany, some understanding of wind currents. There is left the understanding of human nature, and the cultivation of its intelligence to secure a concerted campaign in eradication. The enthusiasm of health resorts or towns and the zeal of local service clubs may be commended as examples of public spirit, but must be well advised to secure co-operation in factual result.

Air pollution by ragweed pollen is a menace to public health. Should not its destruction become a proper function of the state health bureau? Would not each householder or tenant destroy his own weeds rather than have the health board direct public attention to the nuisance? Destruction is advised prior to August 1. If neglected, should not health officers be empowered as with other health nuisances to destroy such pests at the expense of the owner? A sentiment can be built up which would make the public ragweed-conscious, and which would discredit neglect in its destruction.

SUMMARY

Owing to the geographical position of the state, with its more or less protective boundaries against ragweed, Maine should be a good proving-ground for a concerted campaign of eradication. A pollen survey was first necessary. Beginning August 8, 1937, slides were exposed for each of fifty days at nine authorized receiving stations in cities, farming towns, mountains and seashore, and were carefully counted. Maps, graphs, and tabulations accompany the text. Air currents as distributing agents were studied. Weather bureaus and forest wardens contributed to the success of the survey in furnishing data of storms, wind directions and velocities. A low ragweed count was found in nearly the whole state, a negligible amount in agricultural Aroostook County; the highest count, comparable with that of neighboring states, was in the southwest corner, which does not have any mountain-forest barrier.

Ragweed pollinosis is rated as an air-pollution problem for boards of health. An appeal is made for intelligent state-wide co-operation in eradication.

REFERENCES

1. Durham, O. C.: Evaluation of ragweed hay fever resort areas of North America. J. Allergy **8**:175-189, 1937.
2. *Idem:* Incidence of ragweed pollen in United States during 1929. J. A. M. A. **94**:1907-1911, 1930.

DIVERTICULUM AND SARCOMA OF THE DUODENUM

Report of a Case

ANTHONY J. MENDILLO, M.D.,* AND WILLIAM B. KOUFMAN, M.D.†

NEW HAVEN, CONNECTICUT

DIVERTICULA of the duodenum have been recognized with greater frequency since x-ray diagnosis has become a major part of our clinical regime. Friedlaender‡ states that radiologists have found them in every third case of "gastric disturbance," and pathologists in up to 5 per cent of their postmortem examinations. There have also been 62 cases of sarcoma of the duodenum reported in the literature, and they have presented a wide variety of clinical pictures. The case here reported, however, represents a combination of both diverticulum and sarcoma of the duodenum which we believe is the first case of its type to be described.

CASE REPORT

V. T., a 49-year-old Lithuanian, married man, with an essentially negative past history, was admitted to the hospital on October 11, 1937, complaining chiefly of weakness.

He was well until 3 years before admission, when he first became disturbed by recurrent episodes of epigastric distress about an hour after eating. This vague discomfort was not relieved by further taking of foods or alkalies. Coincident with the onset of this symptom he began to notice that his stools were tarry, that he became fatigued very easily, and that he was slowly becoming weaker. An x-ray photograph was taken and a report of an "ulcer of the stomach" was given. With this as a basis his physician put him on an ulcer regime with Sippy powders A and B. In 1 month, he stated, he was symptom-free, and remained so until 1 year previously. In this interim of 2 years he maintained his weight, was on an unrestricted diet, and led a normal active existence.

One year previous to admission he again began to notice tarry stools, and within a relatively short time he began to feel weak and fatigued on slight exertion. However, he had no symptoms referable to the gastrointestinal tract. Evidently his complaint of tarry stools was ignored, and on routine examination he was found to be "anemic." After a series of liver injections over a period of 5 weeks he again felt well for a short time. Ten months, 6 months and 1 month previously he had recurrences of melena and weakness. After the last episode he was referred to the hospital. During this year he lost 20 lb.

Physical examination revealed a well-developed, well-nourished man lying comfortably in bed in no pain or distress. The mucous membranes were pale. The cardiovascular and pulmonary systems were essentially negative. The blood pressure was 130/70. The abdomen was relaxed, scaphoid and with a moderate amount of subcutaneous tissue. There were no palpable masses and no tenderness, spasm or rigidity. There were no hernias. Neither liver, spleen nor kidneys were palpable. Rectal examination was normal. The extremities and reflexes were also normal.

Examination of the blood showed a hemoglobin of 50 per cent (Tallqvist). The red-cell count was 2,900,000 and

From the Grace Hospital, New Haven, Connecticut.

*Chief of Staff and of Surgical Division, Grace Hospital, New Haven.

†Resident in surgery, Grace Hospital, New Haven.

‡Friedlaender, G.: Diverticula of duodenum. Brit. J. Radiol. **10**:26-37, 1937.

the white-cell count 5800, with 54 per cent polymorphonuclears, 44 per cent lymphocytes, 2 per cent eosinophils and 2 per cent reticulocytes. There were occasional stippled red cells and some polychromatophylia. There was marked achromia and slight anisocytosis. The blood-sugar content was 90 mg., and the nonprotein nitrogen 32 mg. per cent. Urinalysis was essentially negative and showed a specific gravity of 1.022. The first specimen of gastric contents showed a total acidity of 30, a second specimen one of 20 and a third one of 30. Free hydrochloric acid was absent in the three specimens, but all showed occult blood. There was bile in the feces and a benzidine test was positive. Kolmer and Kahn tests on the blood were both negative. An x-ray film revealed a steer-horn type of stomach, high in position, with an even and regular contour; there were no filling defects. The pyloric portion was well filled and normal in appearance. The duodenal cap was of normal size and shape, but immediately below it was a pocket of barium which was more or less constant on all the radiograms; this was held to be consistent with a diverticulum of the duodenum. The stomach emptied normally; a 6-hour plate showed no gastric residue, but a considerable amount of residue remained immediately below the duodenal cap. The remainder of the gastrointestinal series was essentially negative.

Using pontocaine and novocain for subarachnoid block, the abdomen was opened through an upper abdominal midline incision. The stomach, pylorus and liver were found to be normal. In the region of the duodenum and closely associated with it there was found a moderately soft, rounded mass about the size of a lemon, which was markedly adherent above, below and posteriorly. It was not associated with the pancreas, and so far as could be determined, involved only the second portion of the duodenum anteriorly. The mass was carefully dissected free of the adhesions, and it was then noted to involve and to be directly associated with the second portion of the duodenum anteriorly, in which a double-barreled diverticulum was located. The tumor mass was removed, and the opening in the duodenum thus brought about was sutured transversely with a triple row of intestinal sutures; the area of repair was covered with omentum. A cigarette drain was inserted down to the operative region, and the abdomen was closed in layers.

The patient made an uneventful and uncomplicated recovery and was discharged from the hospital on the 18th postoperative day. X-ray therapy was not advised.

Pathological Report. "This specimen was received as a diverticulum of the duodenum. It measured 10 by 7 by 4 cm. It was grayish in color and irregular on the surface. At one end there was an opening of considerable size, evidently where it had been removed from its connection with the intestine. On cutting into the specimen there was revealed a cavity which communicated with the opening and extended down the length of the specimen but not entirely through it. The inner surface was irregular, the wall quite firm and grayish in color, and varying from 2 to 5 mm. in thickness. In gross it gave the impression of a malignant tumor with cavitation. No mucous membrane was recognized on the inner surface of the cavity. There was some greenish discoloration with a few eroded, reddish patches on the inner surface.

"Sections for microscopical examination were taken from different parts of the wall of the cavity. These show a cellular tumor growth. The cells are of good size, elongated where cut lengthwise, and suggest a tumor originating in non-striated muscle. The inner surface, as shown by most of the sections, consists of tumor growth with superficial necrosis and without any mucous membrane. One of the sections showed intact intestinal mucous membrane. Diagnosis: leiomyosarcoma."

Eight months postoperatively the patient's general condition was excellent. He had been doing hard outdoor work for the previous 3 months. His appetite was excellent, he had been on an unrestricted diet, and he had gained 38 lb. since the onset of his illness. The red-cell count was 5,050,000 and the hemoglobin 95 per cent (Dale). The patient was on no medication and had had no digestive disturbances. Examination revealed a well-healed operative scar. No masses were palpable in the abdomen, and the liver edge could not be palpated. There were no adenopathies. Repeated x-ray study of the stomach and duodenum at this time revealed the descending duodenum to be somewhat irregular, with considerable deformity involving the duodenal cap. There was no retention of barium after 6 hours.

SUMMARY

A case of diverticulum and leiomyosarcoma of the duodenum is reported.

The chief clinical manifestation was that of secondary anemia. Evidently the bleeding occurred in exacerbations and was caused by ulceration of the mucosa of the diverticulum. The diverticulum, but not the tumor, was diagnosed by x-ray. At operation, there was no evidence of metastases in the liver or surrounding tissue.

PROGRESS IN DERMATOLOGY

JACOB L. GRUND, M.D.*

BOSTON

THE most important contributions to the dermatological literature of the past year have been in the field of therapeutics. Though not numerous, they should prove of value in a field of medicine that concerns itself to so great an extent with treatment. Noteworthy also have been the studies of dermatoses in which allergy plays a predominant part. Sulzberger and his associates have aroused considerable interest in this aspect of dermatology, and more has been accomplished by their researches in this field in the last few years than for some time past. The more pertinent of the many articles that have appeared are, however, not entirely limited to the above, but as will be seen by the summaries presented, cover additional interesting phases of dermatology.

The outstanding symptom of any skin disease is undoubtedly pruritus. The exact manner of its production is as yet not fully understood. Aubrun's[1] extensive experimental work in this connection is therefore of value. By the partial denervation of the sensory nerves of animals he was able to produce a hyperesthesia or initial pruritus as evidenced by reflex scratching. The latter symptom was confined to the area supplied by the severed nerve. It is his belief that sectioning of the nerve liberated an algogenic substance or one causing pruritus through its action on the intact sensory nerve endings.

Scleroderma has always been an intriguing subject, not only to the dermatologist but to the neurosurgeon as well. Previous contributions have connected its appearance with some form of involvement of the sympathetic nervous system. Cornbleet and Struck,[2] however, offer the hypothesis that scleroderma is primarily due to the action of a toxin on the collagen syncytium, which secondarily takes up calcium. Both calcium and phosphorus are retained in the body, as is evidenced by the abnormally small quantities of these substances excreted in the urine of patients with scleroderma. The authors found that with large doses of viosterol this balance became negative, as indicated by the marked increase of the calcium content of the urine. It was necessary to administer 200,000 to 300,000 international units of vitamin D (viosterol) for as long as four months in order to obtain improvement. Cases of sclerodactylia were affected by this treatment more slowly. The benefit obtained was undoubtedly gained at the expense of the calcium previously deposited in the collagen and muscles of the involved areas. No mention of the exact nature of the toxin or of its manner of production is made in the article. Furthermore, it should be borne in mind that cases receiving such large amounts of viosterol must be closely watched for possible toxic effects.

Although the common locations of the lesions of lichen planus are well known, the case reported by Milian and Périn[3] indicates unusual possibilities. Their patient, a woman of thirty-three, had complained for several years of vague gastric upsets. Although her skin was free of disease, she presented on the tongue and buccal mucosa lesions which were clinically and histologically typical of lichen planus. On gastroscopic examination, typical lesions of lichen planus were found in the stomach.

During the last three years many references have appeared regarding the etiology of erythema nodosum. Spink[4] studied 10 cases and reviewed the records of 131 additional patients seen at the Boston City Hospital over a ten-year period. In 5 of the first 10 patients the eruption was preceded by a sore throat; in 3 there was a history of an "abscessed tooth" shortly before its appearance. In 133 cases no significant relation to tuberculosis was found. Bacterial and immunological studies suggested a causal relation with *Streptococcus hemolyticus*. The author was able to reproduce lesions histologically identical with those of typical erythema nodosum, and has thus been able to demonstrate clearly the infectious nature of the disease, which has for some time been assumed without proof. Spink concludes that erythema nodosum appears to be a nonspecific inflammatory reaction of the skin to a variety of bacterial, toxic and chemical agents.

Nothing startling has been written during the last year on psoriasis. However, the case reported by Spillmann et al.[5] is of interest. A girl of twelve was first examined in December, 1935, because of a generalized resistant psoriasis and a body habitus suggestive of dystrophia adioposogenitalis. After twelve treatments of short-wave diathermy to the region of the hypophysis, the psoriasis entirely disappeared. In the spring of 1936 it reappeared, and was uninfluenced by anterior pituitary injections and vitamin C. Early in September, 1936, the menses appeared for the first time, and within two

*Instructor in dermatology and syphilology, Boston University School of Medicine; assistant dermatologist, Department of Dermatology and Syphilology, Massachusetts Memorial Hospitals.

weeks the eruption entirely cleared. These authors believe that endocrine factors rather than pure coincidence explain these phenomena.

Gougerot and Degos[6] describe a method of treating psoriasis which they think is less liable to be followed by recurrences than are the usual forms of therapy. A 2 per cent solution of eosin in alcohol is applied to the lesions every two days. On alternate days they are anointed with either petrolatum or cold cream. Between the applications of eosin the patient takes a bath in which has been dissolved 125 gm. of soda bicarbonate. A fat-free diet is prescribed. The eosin has no odor and quickly washes off. The authors maintain that the results are at least as good as those to be obtained with chrysarobin.

The common medical belief of a causal relation between certain cutaneous affections and diet is emphasized by the experiments of Pillsbury and Sternberg.[7] By means of intradermal injections of broth suspensions of living staphylococci and streptococci in dogs, they found that the cutaneous infection produced was more severe in animals on a high-carbohydrate diet than in those with a low-carbohydrate or high-fat intake and than in fasting animals.

Mitchell[8] believes that many of the so-called eczematous eruptions in and around the ears are in reality due to streptococci. In fact the lesions have been termed by Sabouraud *Streptococcides eczématiformes*. Retroauricular intertrigo and scaly exudative patches in the cavum and the canal may be caused by these organisms. Itching is a common symptom. Mitchell claims that streptococci can be readily demonstrated in the serum of the fissures and in the squamous-like crusts which characterize this infection.

One of the most practical and important contributions to dermatology, especially its allergic phase, is that of Goodman and Sulzberger.[9] Dealing with allergic eczematous eruptions or contact sensitivity, they present the various types of eruptions that may occur in patients who contact certain substances. In affected subjects, the lesions are confined to the skin and are characterized by the definite histologic findings of spongiosis or intraepidermal vesicle formation. The sensitivity present is nonprotein in character and has no association with asthma, hay fever, rhinitis or infantile eczema. It is not familial.

The commonest form of this disorder is dress dermatitis. In two years Goodman and Sulzberger saw 25 cases. They found that most of the patients had hyperidrosis, particularly of the axillas. The eruption affected the axillary and periaxillary regions but spared the pit. This fact differentiated it from moniliasis, seborrheic dermatitis and psoriasis. The antecubital spaces and sides of the neck were usually also involved. In the more severe cases, the arms, thighs, buttocks and in fact the entire body often presented changes. But in no case did the eruption spread to the hands, feet or lower legs. The typical features were erythema, edema, papulation and vesiculation. The pigmented, thickened, lichenified areas or the large excoriated papules of atopic dermatitis were never seen. In all the cases the patch test was positive. The authors believe that the dyes used in the dresses were the probable cause of the dermatitis. Removal of such clothing and the application of antipruritic lotions and roentgen-ray treatment usually cleared the disorder.

Another example of contact sensitivity described is shoe dermatitis. This appears to be comparatively rare. However, the fact that it may easily be confused with ringworm warrants a description of its essential features. Patch tests indicate that inner linings, brown and black uppers and even black and brown shoe polishes may be responsible for its presence. The eruption is eczematous in character, and its symptoms are erythema, scaling and vesiculation, features that are quite characteristic of dermatophytosis. The following features differentiate it from the latter: (1) in most cases there is little or no involvement of the interdigital spaces and little or no maceration; (2) the juxtaposed areas of the toes are not involved; (3) there is a characteristic involvement of the dorsums of the toes, particularly the great toes; (4) the eruption is most marked at the site of the maximum pressure or friction from the shoes; (5) there is a tendency for the eruption to be demarkated by the shoe margins; (6) procedures usually successful in treating dermatophytosis prove unsatisfactory; (7) the itching is more severe than is usual in uncomplicated dermatophytosis. The last, and more important type of dermatitis taken up by Goodman and Sulzberger is that caused by cosmetics. It is their opinion that its occurrence is due to a sensitivity to the pigments in powders, creams, lotions, dentifrices, rouges and lipsticks. The indelible dyes of the bromofluorescin group constitute the most frequent cause. Many cases are caused by the nonindelible lake colors, in particular 1-sulfo-β-naphthalene-azo-β-naphthalol. The fatty ingredients and perfumes do not produce a dermatitis.

Caulfield[10] discusses in more detail the differences between a contact and an atopic dermatitis. The modus operandi in the contact group is actual contact with the offending substance, whereas the atopic lesion is caused by its inhalation or ingestion. The excitant in the contact group is found in the ether-soluble fraction of the agent; in the

atopic group it is present in the water-soluble or antigenic fraction. In the contact cases, heredity exerts no effect; the patch test is positive and the intradermal negative; skin-sensitizing antibodies are absent in the serum, as evidenced by the Prausnitz-Küstner reaction. In the atopic cases, the factor of heredity, as evidenced in hay fever and asthma, plays an important part; the intradermal test is positive, and the patch test negative; the Prausnitz-Küstner reaction, obtained by applying a patch-test to a serum-sensitized site, is positive. Caulfield describes a case with a history of seasonal exposure for four years to tulip bulbs which resulted in a severe dermatitis. A prolonged dermatitis followed the application, in the form of a patch test, of the ether-soluble portion obtained from minced tulip bulbs. Intramuscular injections of the ether soluble fraction (non-antigenic) taken up in oil (Mazola) prevented a dermatitis for the first time in five years.

Of value is the report by Sternberg[11] of cases of contact dermatitis caused by oil of cloves and camomile tea. Both these substances are used extensively, the former in various cosmetics, drugs, candies, tooth powder and shaving soaps, and the latter as a hair dye. Patch tests with the suspected substances in these cases were positive.

Although other substances, among them bismuth and manganese, are being employed in the treatment of lupus erythematosus, many dermatologists still adhere to the method of injection with gold. An interesting case[12] is that of a twelve-year-old boy whose lupus erythematosus started after a sun bath. Definite improvement occurred with injections of a preparation made up of 12.5 per cent gold and a derivative of a sulfur protein obtained from skin keratin and supporting tissue. The same preparation was used with good effect in a patient with rectal stricture due to lymphgranuloma venereum. That gold is not entirely innocuous is emphasized by the report of Irgang.[13] His case was that of a chronic lupus erythematosus, which after the eighth injection of a small dose of a gold compound presented a melanoderma and after the tenth dose, a keratoderma and mild stomatitis. It should further be borne in mind that the injection of gold salts may produce an extreme and indeed fatal degree of aplastic anemia.

Preparations of gold have been effectual in the treatment of erythema induratum or Bazin's disease. Glaze,[14] however, describes a much simpler method. He obtained complete involution and relief after the daily application for three weeks of a mixture made up of equal parts of collodion (U.S.P.) and flexible collodion (U.S.P.). Repeated applications, each over the previous one, were made on the affected areas and the sound skin surrounding them.

In a previous progress report, Hruzek's method of treating recurrent herpes simplex by means of autoinoculation was described. Foster and Abshier[15] have reported a method of treatment which has been used in Europe for several years. Thirty-five cases of this comparatively insignificant yet troublesome eruption were inoculated with smallpox vaccine. After two years, there were only 14 per cent recurrences.

Nothing startlingly new has appeared in the literature regarding the treatment of pemphigus. Reports[16] appear from time to time abroad of the excellent results obtained with Germanin. This drug, however, has proved ineffectual in the hands of American dermatologists.

Goldman[17] writes of a new treatment for scabies. It has been used in Denmark for a number of years and has replaced sulfur ointment. It possesses certain advantages over the latter in that it is odorless and non-irritating. The procedure is as follows: The patient takes a hot bath, after which a solution made up of benzyl benzoate 50 gm., liniment of soft soap 65 gm., alcohol (90 per cent) 30 gm., and distilled water 5 gm. is applied over the entire body with a small brush and allowed to dry. Another layer is then applied in similar fashion. After the skin is dry the patient dresses. After another bath the next day he changes to fresh clothing. The same precautions are taken in laundering of clothing as are observed in the other methods of treatment.

The problems of cancer are still with us, and in this connection the observations of Sachs and Garbe[18] should prove of interest. The textbooks teach us that basal-cell epithelioma occurs only rarely on the extremities, and that certain of its features, for instance the so-called raised, pearly border, are pathognomonic. The authors list 32 cases in which a basal-cell epithelioma was present on the extremities. As is usually the case, they progressed very slowly and almost never metastasized. In many cases, however, they closely resembled prickle-cell cancer and could be differentiated from it only by pathological examination.

Following the recent work of Lawrence with a glandular preparation in the treatment of acne, it was thought that a more logical method of treating this common skin disease than with the roentgen ray was at hand. Authoritative opinion among dermatologists is to the effect that there is an etiologic connection between a gland or glands of internal secretion and acne vulgaris. They have nevertheless been unable to substantiate the results obtained by Lawrence. An article by McCarthy and Hunter[19] clearly indicates this. Their results in

the treatment of acne with gonadotropic substance from the urine of pregnant women and Theelin were distinctly disappointing. With the work of Tanenbaum et al.[20] with staphylococcus toxoid as a foundation, more promising effects in the treatment of this disease seem possible. Of 100 cases that received toxoid, excellent results were obtained in 9 per cent, good results in 49 per cent, some improvement in 37 per cent and no change in only 5 per cent. Its best effect was in pustular acne. In the discussion of this article, Sulzberger and Rubin[21] describe their observations in the treatment of acne with this substance. Twenty-five cases both of the vulgaris and of the cystic type were followed for two years. All the cases had proved resistant to other treatment. Every one of 6 cases of cystic acne in older women was benefited. Three cases of acne varioliformis cleared rapidly and strikingly. It appears from these results that the toxoid is a worthwhile adjuvant to the roentgen ray in the management of this condition. The preparation was employed also in 5 cases of sycosis vulgaris resistant to all other forms of therapy. Two were completely cured, 2 were greatly improved, and in 1, treatment was a complete failure. Three cases of hydroadenitis axillaris were rapidly cleared. Using the toxoid, Whitby[22] obtained a good clinical effect in a high proportion of patients with recurrent and resistant furunculosis. He found the preparation useful in the treatment of styes and carbuncles.

Reports regarding the value of the roentgen ray in infections continue to appear. Hodges and Berger[23] consider it an important adjunct to other remedies in the management of carbuncles and certain fungus infections (blastomycosis and sporotrichosis). They found the following conditions amenable to the x-ray without other therapy: early localized erysipelas in adults, furuncles and furunculosis, granulomas, infected hemangiomas, certain types of cellulitis and lymphangitis, Mikulicz's disease, parotitis and rhinophyma. They noted that the more acute the infection, the smaller was the dose of roentgen rays required. The process of destruction was hastened, the result being more profuse drain of the treated area. These changes, they believe, are based on the sensitivity of the leukocytes to the rays, liberation of ferments, stimulation of antibodies and electrocolloidal and metabolic changes in the treated cells.

Despite the fact that cutaneous roentgen-ray therapy is standardized to the extent that untoward reactions are avoidable, they nevertheless do occur. Overexposure and too long-continued treatment are the prime causes of their presence. The resulting dermatitis and subsequent ulcer formation usually prove debilitating and serious, especially from the standpoint of possible future cancer formation. McLaughlin's[24] report of Kesten's treatment of this complication is therefore interesting. In this case the patient presented on both hands telangiectasia, keratoses, crusting and fissuring as a result of sixty one-quarter erythema doses administered from 1925 to 1931. Following the use of an 8 per cent viosterol ointment, the condition was markedly improved; the crusting disappeared, and many of the keratotic lesions were no longer visible and in certain areas were greatly reduced in size. Loveman[25] recommends the leaf of aloe vera, applied both in the form of the fresh whole leaf and as an ointment, to the roentgen-ray ulcer. He found the leaf more effective than the ointment. The latter was fairly satisfactory for the keratoses and for improving the general tone and texture of the skin. Continuance of the treatment is advised for at least three to nine months before it is given up. It was observed that the younger the ulcer, the more effective was the treatment.

475 Commonwealth Avenue.

REFERENCES

1. Aubrun, E. A.: Pruritus and hyperesthesia caused by partial sensory denervation; experimental alopecia — contribution to study of alopecia areata. Arch. Dermat. & Syph. **34**:564-567, 1936.
2. Cornbleet, T., and Struck, H. C.: Calcium metabolism in scleroderma. Arch. Dermat. & Syph. **35**:188-201, 1937.
3. Milian, G., and Périn, L.: Lichen plan gastrique. Bull. Soc. franç. de dermat. et syph. **43**:644-647, 1936.
4. Spink, W. W.: Pathogenesis of erythema nodosum with special reference to tuberculosis, streptococcic infection and rheumatic fever. Arch. Int. Med. **59**:65-81, 1937.
5. Spillmann, L.; Drouet, P. L., and Weille, R.: A propos d'un psoriasis blanchi des l'apparition de la puberté. Bull. Soc. franç. de dermat. et syph. **44**:22-23, 1937.
6. Gougerot, H., and Degos, R.: L'eosine dans le traitement local du psoriasis. Bull. Soc. franç. de dermat. et syph. **43**:1784-1785, 1936.
7. Pillsbury, D. M., and Sternberg, T. H.: Relation of diet to cutaneous infection. Arch. Dermat. & Syph. **35**:893-909, 1937.
8. Mitchell, J. H.: Streptococcic dermatoses of the ears. J. A. M. A. **108**:361-366, 1937.
9. Goodman, J., and Sulzberger, M. B.: Acquired specific hypersensitivity to simple chemicals; eczematous sensitivity to clothing and to cosmetics; with special reference to dyes. J. Allergy **9**:136-157, 1938.
10. Caulfield, A. H. W.: Report of the investigation and successful treatment (preventive) of dermatitis resulting from the handling of tulip bulbs. J. Allergy **8**:181-184, 1937.
11. Sternberg, L.: Contact dermatitis; cases caused by oil of cloves and by oil of camomile tea (anthemis cotula). J. Allergy **8**:185-186, 1937.
12. Pillokat, A.: Ein ungewöhnlicher Fall von Lupus erythematodes generalisatus, zugleich ein Beitrag zur Therapie des Lupus erythematodes und anderer Dermatosen mit Auro-Detoxin. Dermat. Wchnschr. **102**:193-199, 1936.
13. Irgang, S.: Keratoderma and melanoderma accompanying therapy with a gold compound; report of a case. Arch. Dermat. & Syph. **34**:624-629, 1936.
14. Glaze, A. L.: Treatment of erythema indurata (Bazin's disease). Arch. Dermat. & Syph. **34**:888, 1936.
15. Foster, P. D., and Abshier, A. B.: Smallpox vaccine in the treatment of recurrent herpes simplex. Arch. Dermat. & Syph. **36**:294-301, 1937.
16. Donges: Über Germaninbehandlung bei Pemphigus vulgaris und foliaceus unter besonderer Hervorhebung eines atypisch verlaufenen Falles. Dermat. Wchnschr. **102**:277-282, 1936.
17. Goldman, L.: Treatment of scabies with benzyl benzoate. Arch. Dermat. & Syph. **36**:140, 1937.
18. Sachs, W., and Garbe, W.: Primary basal cell epithelioma of the extremities; review of literature and report of 32 additional cases. Arch. Dermat. & Syph. **36**:273-278, 1937.
19. McCarthy, L., and Hunter, O. B.: Failure of therapy with glandular preparations in acne vulgaris. Arch. Dermat. & Syph. **35**:211-225, 1937.
20. Tanenbaum, S.; Joyner, A. L.; Speed, J. A., and Bremer, K. M.: The use of staphylococcal toxoid in the treatment of 100 cases of acne vulgaris. J. Allergy **9**:241-245, 1938.
21. Sulzberger and Rubin: discussion of paper by Tanenbaum et al.[20]
22. Whitby, L. E. H.: The treatment of staphylococcal skin lesions with toxoid. Lancet **1**:1454-1456, 1936.
23. Hodges, F. M., and Berger, R. A.: Roentgen therapy of some infections. J. A. M. A. **107**:1551-1555, 1936.
24. McLaughlin, R.: Roentgen ray dermatitis treated with ointment containing viosterol. Arch. Dermat. & Syph. **34**:901-903, 1936.
25. Loveman, A. B.: Leaf of aloe vera in treatment of roentgen ray ulcers; report of 2 additional cases. Arch. Dermat. & Syph. **36**:838-843, 1937.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24381

PRESENTATION OF CASE

First admission. A sixty-two-year-old Italian laborer entered the hospital with the complaint of shortness of breath of six months' duration.

He was essentially well until about six months before entry, when he suddenly had a severe shaking chill which lasted for about five hours. Following this he perspired profusely. He remained in bed for two weeks and felt reasonably well except for severe dyspnea. He was so dyspneic that he was unable to lie flat in bed. Because of this he was sent to an outside hospital where a diagnosis of pneumonia was made. He remained there about six weeks but had no cough, sputum, pain in the chest or other symptoms except the dyspnea. When he returned home, he was unable to work because of severe dyspnea on the slightest exertion. Exertion also brought on regular palpitation of the heart. About two months before entry he began to have in both shoulders constant nonradiating pain which interfered somewhat with his sleep and was more severe when he sat up. Six weeks before entry he began to notice that he was less energetic than usual and found that he had lost about 30 lb. in weight. For some time his appetite had been rather poor. Two weeks before entry his physician removed about sixteen ounces of wine-colored fluid from his right chest. This gave him some relief from symptoms. One week before entry about 40 oz. of similar fluid was removed. To his knowledge he had no further fever after the acute onset of his illness and never any cough, sputum, hemoptysis, gastrointestinal or genito-urinary symptoms. His past and family histories were essentially negative.

Physical examination revealed a well-developed, fairly well-nourished man sitting up in bed in no great discomfort. The fundi showed arteriovenous nicking. The lungs showed dullness to flatness at the right base, with increased resonance over the left lung. Breath sounds and voice sounds were diminished over the upper part of the right lung and absent at the right base and increased over the left lung, and wheezes could be heard in it. The mediastinum and heart were shifted to the left. The apex impulse could be felt 12 cm. to the left of the midsternal line in the sixth interspace. There was a harsh systolic murmur heard best at the apex. There was a pulsus alternans, and the blood pressure was 155 systolic, 130 diastolic. The abdomen was negative.

The temperature was 99°F., the pulse 90. The respirations were 20.

Repeated urine examinations were negative. The blood showed a red-cell count of 4,830,000 with 75 per cent hemoglobin, and a white-cell count of 12,150 with 87 per cent polymorphonuclears. Four sputum specimens were negative for tubercle bacilli, and the stool was guaiac negative. The blood Hinton test was negative. The corrected sedimentation rate was 0.8 mm. per minute, a Takata-Ara test was negative, and a 1:20,000 tuberculin test was positive. The nonprotein nitrogen of the serum was 27 mg., the calcium 10.03 mg., the phosphorus 4.40 mg. and the serum protein 5.89 gm. per cent. The van den Bergh was normal. The chlorides were equivalent to 103 cc. of N/10 sodium chloride. A bromsulfalein test of liver function showed 0.5 per cent retention.

An x-ray taken one week before entry showed dullness at the right base obscuring the outline of the diaphragm. In the lateral view the right lower lobe was seen to be much decreased in size. Part of the dullness at the base was due to a rounded shadow extending along the interlobar septum between the upper and middle lobes. The upper lobe was increased in size and radiance, and, immediately beneath the sternum in the anterior portion of the chest, there were irregularly rounded areas of rarefaction suggestive of bleb formation. On the left side the costophrenic angle was obliterated, and there was thickening of the axillary pleura. The heart shadow was increased considerably in size, the enlargement being in the region of the left ventricle. The aorta was tortuous, and there was calcification in the arch. An x-ray taken after the first chest tap in this hospital showed homogeneous dullness on the right side rising to the level of the eighth rib posteriorly and ending in a sharp horizontal line, probably due to air above it. The apex of the right lung was separated from the chest wall by air, and the right lower lobe was partially collapsed. The heart and mediastinum were slightly displaced toward the right. The left lung was clear. An x-ray of the abdomen showed homogeneous dullness in the upper portion which was thought to be due to a moderately enlarged liver.

An electrocardiogram showed moderate left-axis deviation, with late inversion of T_1, low T_2, elevated ST_4 and small R_4.

On the second day 510 cc. of light amber-colored

fluid was removed from the right chest. It had a specific gravity of 1.014, and contained 5040 cells with 58 per cent polymorphonuclears, 30 per cent lymphocytes and 12 per cent eosinophils. No tubercle bacilli could be identified in it, and a guinea-pig inoculation failed to produce tuberculosis. A culture gave bacillus coli and nonhemolytic streptococci. Histological examination showed no tumor cells. Bronchoscopy on the fifth day showed no evidence of tumor or obstruction. A second chest tap yielded 1475 cc. of similar fluid, and x-rays taken after that showed that most of the fluid had been removed. The right border of the heart and the right lung root were fairly distinct, and the entire right lung was aerated. There was no evidence of tumor. One of the films showed calcification in the region of the aortic valve. The patient remained in the hospital for six weeks and symptomatically showed gradual improvement. For the first three weeks he showed a daily rise in temperature to 100°F., and throughout his stay in the hospital his white-cell count varied between 12,000 and 15,000 with one count of 21,700 and a count of 13,000 on the day of discharge. A third chest tap was done on the thirteenth day. Sixty cubic centimeters of fluid was obtained which had a specific gravity of 1.015. The fluid was clear and was said to contain 120,000 red cells and 25,000 white cells. On culture it gave no growth aerobically or anaerobically. It contained 2.5 gm. per cent protein. On the twenty-second day a fourth tap yielded 410 cc. of clear amber fluid with a specific gravity of 1.015 which contained 1836 red cells and 1629 white cells with 60 per cent lymphocytes and 40 per cent polymorphonuclears. Cultures again were negative, no tubercle bacilli were seen on smear and the total protein was 3.3 gm. per cent. Physical examination on the fifteenth day showed some tenderness in the right costovertebral angle. The tenderness was never marked and gradually subsided. An intravenous pyelogram was done which showed normal excretion and no evidence of disease in the kidneys. The blood culture on the thirty-second day gave no growth. An electrocardiogram on the eighth day showed left-axis deviation, late inversion of T_1 and inversion of T_4. Digitalization was begun on the nineteenth day. An electrocardiogram five days before discharge showed a ventricular rate of 60, inverted T_1, low diphasic T_2, diphasic T_3, inverted T_4 and upright QRS_4. His vital capacity gradually increased from 2.4 liters to 3.0 liters. Several x-rays showed no significant changes except decrease in the amount of the fluid. He was discharged on the forty-third day.

Second Admission (five months later). During the interval his general condition remained about the same. However, his dyspnea gradually increased, and three and a half months after discharge a chest tap was done yielding 1100 cc. of cloudy amber fluid having a specific gravity of 1.010, and containing 11,000 red cells and 3.3 per cent protein. Aerobic and anaerobic cultures again gave no growth, and no tumor cells were seen on histological examination. Two weeks later an x-ray of the chest showed that the hilus shadows had increased in size. The heart was larger, and there were increased amounts of fluid in the right pleural cavity. One month later, a week before re-entry, another tap was done and yielded 1200 cc. of reddish amber fluid. This gave relief for two days but after that his dyspnea rapidly increased, and he became steadily weaker. For about four months before re-entry he had been more constipated than usual but had no nausea, vomiting or bloody or tarry stools. Anorexia was marked.

Physical examination revealed him to be poorly nourished and obviously chronically ill. He was moderately dyspneic and orthopneic. The right chest did not expand so well as the left, and there were dullness and decreased tactile fremitus and breath sounds in the lower right chest posteriorly. The heart was enlarged, and the sounds were distant. There was a loud blowing systolic murmur at the apex. The blood pressure was recorded as 155 systolic, 145 diastolic.

The temperature was 98.6°F., the pulse 85. The respirations were 25.

The urine examination was negative. The blood showed a red-cell count of 4,250,000 with 85 per cent hemoglobin and a white-cell count of 10,400 with 50 per cent polymorphonuclears. The blood Hinton was negative, and the nonprotein nitrogen was 29 mg. per cent. The corrected sedimentation rate was 0.9 mm. per minute. An electrocardiogram showed less deep inversion of T_1 than on the previous record, inversion of T_4 and high ST_3. An x-ray of the chest showed no appreciable change in the appearance of the heart. The amount of fluid in the right pleural cavity had increased, and there was fluid in the interlobar septums. The hilus shadows were wider than normal but not definitely wider than on the previous examination.

He improved somewhat for the first week, but on the evening of the eighth day he was found comatose and in great respiratory distress. He showed Cheyne-Stokes respiration, and the blood pressure was 180 systolic, 140 diastolic. The heart was unchanged, but the reflexes were all hyperactive, and there were bilateral Babinski signs. A lumbar puncture showed an initial pressure of

190, with normal dynamics. The spinal fluid was normal. The following day the nonprotein nitrogen of the serum was 77 mg., and the carbon-dioxide combining power 59.5 vol. per cent. He died the next day with a terminal rise in temperature to 108°F.

Differential Diagnosis

Dr. Walter Bauer: Was this patient well prior to the onset of the present illness?

Dr. Benjamin Castleman: He was so far as we know.

Dr. Bauer: Did he have dyspnea and fever from the very onset?

Dr. Castleman: Apparently he had some fever at the other hospital, where a diagnosis of pneumonia was made.

Dr. Bauer: So you think he probably had fever from the onset?

Dr. Castleman: Yes.

Dr. Bauer: Do you think it persisted for three or four weeks?

Dr. Castleman: I do not know. There is nothing further in the record.

Dr. Bauer: I doubt if one diagnosis will adequately explain all the symptoms and signs encountered in this case. I find it extremely difficult to put this puzzle together. The onset was characterized by a severe chill which lasted five hours. This type of onset is extremely rare or unheard of in connection with some of the diagnoses one would like to consider and discuss. I shall assume that he was dyspneic from the onset. The severe shaking chill must represent entrance into the blood stream of foreign matter or bacteria. If we limit ourselves to diseases characterized by chill, fever and dyspnea at the onset the diagnostic possibilities are also limited.

These three symptoms are common in pneumonia from the onset. However, the subsequent events are not. I doubt if he ever did have pneumonia. If he did have, there is a possibility that he may have had a complicating interlobar empyema. Such a diagnosis seems unlikely because of the cytological findings in the pleural fluid.

One might entertain the possibility of pericarditis from the very onset of his present illness. This diagnosis is difficult to substantiate, however.

He may have had an initial bacteremia or initial septicemia, the cause of which is not ascertainable from the data presented. It evidently did not persist. Therefore I doubt if its presence has to be entertained seriously. One wonders whether the initial chill, fever and marked dyspnea might have been the result of multiple infected pulmonary emboli. Such a state of affairs would explain his initial clinical picture. If one assumes that such was the case one next has to establish where these infected emboli came from. Certainly chills and fever are rarely associated with non-infected pulmonary emboli. Therefore one is inclined to believe that we are dealing with infected pulmonary emboli.

Spontaneous pneumothorax may manifest itself with the symptom of acute dyspnea. I do not believe that this possibility has to be entertained very seriously, whether it be part and parcel of a tuberculous process or the result of rupture of an emphysematous bleb.

A true chill followed by fever is rarely the first symptom of tuberculosis. This clinical story is not that of miliary tuberculosis. Diffuse miliary tuberculosis often causes marked cyanosis, but rarely is the patient dyspneic. The patient can usually lie flat in bed. I do not think that this man had tuberculosis.

Any number of diseases of the mediastinum give rise to the symptom, dyspnea. However, diseases such as hemorrhage into the thyroid, thymoma, hemorrhage into the thymus and hemorrhage into a mediastinal tumor are not associated with chills and fever. No one of these diagnoses need be considered. The same is true of such diseases as a dissecting aneurysm or rupture of the aortic valve. He had no pain at the onset. Could he possibly have had a mediastinitis? He may have. Certainly it is impossible to establish the cause, and the subsequent story is not a good one for such a diagnosis.

In reading this record over I could not get away from the fact that we were dealing with an individual suffering from several diseases. He may have suffered from pulmonary embolism. Such embolism may have been the cause of thrombosis of the pulmonary artery. On the other hand, that is not sufficient to explain everything that this patient complained of. We have to make some other diagnosis in addition.

Was he cyanotic when he entered the hospital? If so, was the cyanosis associated with engorgement of the neck veins and enlargement of the liver? I suppose the fact that these findings were not mentioned in the record means they were not present.

I should prefer to explain the pain in the shoulders as being due to bilateral bursitis rather than being directly related to the present illness. The fact that this shoulder pain was worse when he sat up means nothing to me.

As we go through this record there are certain things that stand out. He had a harsh systolic murmur best heard at the apex. Nothing is said about what it sounded like at the base, or whether it was transmitted to the neck vessels

and various other facts that might be helpful. When he entered the hospital the first time pulsus alternans was present and the blood pressure was 155 systolic, 130 diastolic. This I interpret as meaning a weak left ventricle in an individual who probably had a pre-existing hypertension. This same finding may, of course, be associated with a pericardial effusion, except that the diastolic pressure is too high. If this man had had a pre-existing hypertension, this finding would suggest a recent coronary occlusion. This is another diagnosis which must be considered.

It is confusing to find that four sputum examinations were made, although it was repeatedly emphasized that no sputum was ever raised. His white-blood-cell count and sediment rate suggest the existence of infection. However, such findings might be accounted for in other ways. Why all this laboratory work was done remains somewhat of a mystery to me. From the case record one would gather that disease of the liver had been suspected. Why, I do not know, because the liver was never palpated and there were no symptoms referable to it. It was reported enlarged by the roentgenologist. Why the serum calcium, phosphorus and other determinations were made, I do not know. They are of no diagnostic aid.

From the roentgenograms taken elsewhere it was concluded that collapse of the lung (lower right lobe) with a small effusion was present. Here again there is a discrepancy in that the physical findings disagree with such an interpretation because the mediastinum and heart were shifted to the left. I would interpret the physical findings as being consistent with either a pleural effusion or collapse. If it were the latter, we would expect the heart to be shifted to the right rather than the left as herein stated. Later roentgenograms were interpreted as showing a shift of the heart and the mediastinum to the right. These later roentgenograms also suggest the possibility of interlobar fluid, interlobar empyema or simple compression. They do mention the existence of blebs. I cannot believe that he had a pneumothorax subsequent to rupture of an emphysematous bleb. This is not a good history for it, and he never had pain. The initial chill and fever do not occur with pneumothorax of this type. Nor did anyone ever hear the characteristic crunching noise often heard in cases with a ruptured emphysematous bleb. The aspirated fluid did not contain bacteria, except on one occasion when they did find *Bacillus coli* and nonhemolytic streptococci. All subsequent bacteriological examinations were negative. It is obvious that the fluid was a transudate.

Will Dr. Hampton show the x-rays? I have interpreted the situation as follows: a man with hypertensive heart disease and superimposed acute episodes. These episodes may have been due to both coronary occlusion and pulmonary embolism. Following this he has suffered from cardiac decompensation. This I believe explains all that happened in this case.

Dr. Aubrey O. Hampton: This film was taken about one month before the first. This area of density described at the right base is the typical picture of fluid with interlobar extension, not an encapsulated empyema. At that time the heart seemed to be displaced a little to the right and enlarged in the region of the left ventricle. The trachea is in normal position and well visualized. There is no evidence of pressure on the trachea or a mass in the upper mediastinum. The displacement of the mediastinum could be due to tapping and removal of fluid just before the film was taken. We get that picture regularly. It does not mean that there is any primary lesion in the bronchus. The lung merely remains small after the fluid has been removed.

Dr. Bauer: Would you say he had fluid in the left chest?

Dr. Hampton: No, not at this time; but there is thickened pleura along the left axillary line and in some of these other films there are transverse areas of queer linear density not usually seen on the left side because there are normally no transverse interlobar fissures. On the right side such linear density in the region of the septum between the middle and upper lobe is common. Here on the left we have two lines which are unusually thick and short. We believe that these changes are commonly due to healed infarcts. They could be due to an anomalous lobe on the left side with thickened interlobar pleura. The shadows described as blebs are too indefinite to be certain of. This shadow was thought to be calcium in the aorta and it looks like it. The calcification in the region of the aorta is quite plain and extensive in the lateral view, but I do not see it in the anteroposterior film and I cannot be sure that it is in the region of the aortic valve. Certainly the aorta is calcified down to the region of the valve. The films following these show the chest after withdrawal of fluid, reaccumulation of fluid and replacement of fluid with air. Finally we have an opportunity to examine the right lower lobe and we find another of these dense thick lines similar to the one on the left. I think this change in the size of the lung root is perhaps

due to pleural disease near the heart on the right, and possibly due to dilatation of the pulmonary vessels.

DR. BAUER: Was it absent in the next film?

DR. HAMPTON: Yes, it may be shifting fluid rather than actual change in the lung root.

DR. BAUER: Would you say that the heart was appreciably larger now than at the time of the first film?

DR. HAMPTON: Yes, I think the last examination shows a larger heart than the first one.

DR. BAUER: Is there anything about the heart shadow to suggest pericardial effusion?

DR. HAMPTON: No, it looks like left ventricular hypertrophy and calcification of the aorta. Whether the aortic valve is calcified I do not know. This abdominal film is not very impressive. The kidneys are not displaced and I cannot be sure that the liver is enlarged.

DR. BAUER: The best diagnosis that I can make is: hypertensive-coronary heart disease, multiple pulmonary infarcts, question of old and recent coronary thrombosis, arteriosclerosis, probably calcification of the aortic valve and cardiac decompensation. I appreciate that some of these things are not well substantiated by the record, but I do not know how to interpret some of the omissions. Therefore I am going to interpret them as I think best. I do not believe that this man had any serious pulmonary disease other than infarction, nor do I think that any of the other possibilities which I have suggested in the discussion have to be considered very seriously. I should like to hear some further discussion.

DR. WILLIAM D. SMITH: I think it is surprising that he had a five-hour chill as a result of embolism.

DR. BAUER: That does not seem right unless one presupposes that the emboli were infected.

DR. PAUL D. WHITE: The aortic stenosis might help to explain the low pulse pressure in spite of hypertension.

DR. WYMAN RICHARDSON: Is not the considerable reaccumulation of fluid without evidence of cardiac failure elsewhere rather peculiar?

DR. BAUER: Yes, but nothing is said about distended neck veins, enlarged liver or leg edema. I grant you if such omissions mean that they were not present, then it seems unlikely.

DR. RICHARDSON: I should like to entertain the diagnosis of tuberculosis as a possibility.

DR. BAUER: What kind? Do you mean pulmonary tuberculosis with pleural effusion?

DR. RICHARDSON: I was thinking of miliary tuberculosis.

DR. BAUER: I should think anyone who had dyspnea of this degree due to miliary tuberculosis would be cyanotic.

CLINICAL DIAGNOSES

Hypertensive and coronary heart disease with mild left ventricular failure and right pleural effusion.
Cerebrovascular accident.
Bronchopneumonia.

DR. BAUER'S DIAGNOSES

Hypertension.
Arteriosclerosis.
Hypertensive coronary heart disease.
Multiple pulmonary emboli (infected).
Old and recent coronary thrombosis?
Calcified aortic valve.
Cardiac decompensation.

ANATOMICAL DIAGNOSES

Aortic stenosis, calcareous.
Coronary thrombosis, old and recent.
Cardiac infarction.
Cardiac hypertrophy and dilatation.
Pulmonary infarct, healed.
Chronic passive congestion of liver.
Pulmonary emphysema.
Hydrothorax, bilateral, very slight.
Renal infarct.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: Dr. Bauer's impression that no one diagnosis would explain this patient's symptomatology and clinical signs was borne out by the autopsy. Several lesions of major importance were found and each of them must have contributed in some degree to the clinical picture. There certainly was, for one thing, severe heart disease; in fact two serious cardiac lesions were present, a calcified aortic stenosis and an old infarct of the heart based on a thrombotic occlusion of the descending branch of the left coronary. It appeared fibrous and nearly healed although the overlying mural thrombus was not yet completely organized and was evidently the source of a small renal infarct. Since both aortic stenosis and cardiac infarction notoriously tend to lower the pulse pressure, it would be idle to speculate as to which was the primary factor in this instance. Possibly they acted synergistically.

The lungs also showed several important findings. There was an extensive bronchopneumonia, but this can be dismissed quickly as a terminal

event. There was also a significant degree of emphysema, most marked in the right upper lobe but visible also in the left upper lobe. This emphysema taken in conjunction with a band of fibrous scarring in the right lower lobe, to be discussed at greater length in a moment, and which, whatever its pathogenesis, certainly represents destruction of a considerable amount of alveolar tissue, was of sufficient grade to have produced significant pulmonary insufficiency.

Of particular interest to us was the explanation of the various areas of linear density discussed by Dr. Hampton. He mentioned thickening of interlobar pleura and healed infarction as likely possibilities — with the latter more probable on the left side. This patient happened to have a well-developed middle lobe on the left, and no evidence of old infarction on that side. Running completely across the right lower lobe, between the upper and middle thirds, however, was a linear fibrous band forming an almost complete septum between the two parts of the lobe. A microscopic section across this zone shows dense fibrosis with masses of closely packed, tangled elastic fibrils — undoubtedly remnants of the original alveolar walls. Two lesions would produce such an end result, an organized pneumonitis or a healed infarct. In favor of the latter was the finding of a large pulmonary artery a short distance toward the hilus from the zone of scarring. This was completely occluded, except for small canalizing vessels, by an organized thrombus. The occluded portion of the artery lay within normal lung tissue, which would be most unlikely if the thrombosis had been secondary to a pneumonitis. I, therefore, feel confident that we can regard the lesion as a healed infarct of considerable size.

At the time of autopsy there was no significant amount of pleural fluid on either side. We found nothing specific, other than the healed infarct, to account for the pleural effusion demonstrated on the first admission.

Dr. Bauer: Does one have the right to assume that the pulmonary embolus was infected?

Dr. Castleman: I do not know. We are in the process of studying the healing of infarcts but have not progressed far enough to feel that we can distinguish between bland and infected ones in their scarred end stages.

Dr. Alfred Kranes: Do you think the rapid reaccumulation of fluid was due to cardiac failure alone?

Dr. Castleman: Probably.

Dr. Bauer: How big was the liver?

Dr. Castleman: It was only slightly enlarged but showed marked passive congestion on section.

CASE 24382

Presentation of Case

An eight-month-old girl was admitted to the hospital with fever, stiff neck, and a bulging fontanel, of a week's duration. The illness apparently began as an upper respiratory infection, one week after her father had had a sore throat with aphonia, and three days after a sister had had a similar illness lasting only a day.

At the onset of symptoms, one week before entry, the family physician found her throat red and the temperature 107°F. Fifteen grains of sulfanilamide a day were ordered. The following day a lumbar puncture was done because of a bulging fontanel and the fluid found to be cloudy with 2600 cells per cu. mm., mostly polymorphonuclear leukocytes. No organisms were seen on smear or grown from culture. Several lumbar punctures were performed, but no organisms were ever found. The number of white cells fell to 925 per cu. mm. After the first three days sulfanilamide was discontinued because of vomiting, and prontosil, 3 cc. every four hours, was administered. The ears were opened but only serous fluid was obtained. The drums healed rapidly without discharge. The fever was remittent, with the temperature running higher each day. At the end of the week the child was admitted with a diagnosis of aseptic meningitis.

Physical examination revealed an obviously ill infant with a stiff neck and back, bulging fontanel and marked dilatation of superficial scalp veins emanating from the fontanel. The child was alert and responsive. The heart and lungs were normal. Kernig's sign was absent. The ears were slightly crusted, but there was no evidence of inflammation of the drums or mastoid tenderness. The temperature was 103.5°F.

Examination of the blood revealed a red-blood-cell count of 5,000,000 with a hemoglobin of 80 per cent and a white-cell count of 20,700 with 56 per cent polymorphonuclears and 36 per cent lymphocytes. The urine contained no sugar or albumin; but occasional white blood cells were found. The spinal fluid was slightly cloudy and under increased pressure; there were 214 white blood cells per cu. mm. (75 per cent polymorphonuclears); the Pandy test was positive; no organisms were found on smear. The blood Wassermann reaction was negative. X-ray examination of the mastoids was negative.

For the first two weeks in the hospital, the temperature ran a septic course with a swing each day of 3 to 5 degrees, from 98-100° in the morning to 103-105°F. at night. Later the temperature was quite constant at 102°.

Lumbar punctures were done one or two times daily throughout the hospital stay; this seemed especially necessary to relieve the high spinal fluid pressure as evidenced by the extreme bulging of the fontanel. The white blood count in the fluid varied between 214 and 2640 per cu. mm.

The first positive bacteriological finding was reported three days after admission — *Bacillus influenzae* in the blood culture and in the spinal fluid. Subsequent spinal fluid cultures showed *Bacillus influenzae* each time. However, no organisms were found on smears of the spinal fluid until the third hospital week. Thereafter, *Bacillus influenzae* were found on smear at every tap.

Treatment of the child was by intravenous injections of influenza serum, 375 cc. having been given by this means. After positive smears of spinal fluid were obtained, the serum was also given intraspinally together with human blood serum as complement. Prontylin in doses of 7½ to 15 gr. daily was given throughout much of the child's stay in the hospital. (The child's weight was 14½ lb.) An urticaria appeared 6 days after the first intravenous injection. No other disturbing allergic reactions occurred except after one intraspinal treatment when the child went into a collapse, but recovered after Adrenalin injections.

The spinal fluid cultures contained no other organisms than *Bacillus influenzae* at any time. The character of the spinal fluid showed little change, except that the per cent of polymorphonuclears varied between 11 and 75. During the third week, a blood culture was reported positive for pneumococcus, type III. Fluid could not be obtained by lumbar puncture after the twentieth hospital day, and ventricular punctures were then resorted to in order to relieve the pressure. One such tap showed 378 cells, 12 per cent polymorphonuclears, no organisms on smear, .037 per cent sugar, 691 mg. chlorides and 43 mg. per cent protein.

The child died on the twenty-second day in the hospital. The body temperature just before death was 100°F. The nature of the death was that of circulatory and respiratory failure. There was no clinical pneumonia.

Differential Diagnosis

Dr. Richard C. Tefft: I think this history deserves a little comment as we go along. In the first place, the physician who first saw the baby apparently thought he was dealing with an acute upper respiratory infection and I think he was justified in that belief and in ordering treatment as he did. We have no evidence that sulfanilamide produces serious or dangerous effects on the bone marrow in babies and small children, and by far the majority of upper respiratory infections with high temperature are caused by streptococci. Moreover, whatever experimental and clinical evidence we have from the use of sulfanilamide therapy justifies us in the belief that it is most effective if given early in the disease.

"The ears were opened but only serous fluid was obtained." That is frequently a polite way of saying a dry tap. I should think it would help us if we assume serum was obtained and that is an abnormal finding. If anyone opens a normal child's ear, and almost everyone with much experience has done this at least once, there is no fluid and the drum crackles. I do not think one hears the crackle, but the sensation is the same as though one punctured a piece of parchment and we say the drum crackles. In this ear some serous fluid was obtained.

On admission to the hospital several things stand out. There was marked dilatation of the superficial scalp veins. Moderate dilatation would be expected in a baby with meningitis, as this baby obviously had. Also the child was alert and responsive. With influenza-bacillus meningitis a child as a rule is not alert and responsive. The Kernig was negative which does not mean much in babies when the fontanel is not closed. It is more likely to be negative than positive in the presence of meningitis. The red count of 5 million would indicate quite definitely that the baby was dehydrated. Babies eight months old usually have red counts below 5 million anyway, and this child had had a septic process going on for more than a week. That practically always results in a certain degree of anemia and it is hard to account for the high red count except by dehydration, which is a reasonable assumption since she was vomiting considerably.

"X-ray examination of the mastoids was negative." Interpretation of mastoid plates on an eight-month-old baby is a very difficult procedure and these may have been read as negative although there may have been some trouble in them. Nothing is said about the consistence of the spinal fluid, and I should like to know when the atypical gelatinous type of fluid appeared.

Dr. Harold L. Higgins: It did not come until the time of block.

Dr. Tefft: It is worth noting that no influenzal bacilli were found on smear until the fourth week. A child with influenza-bacillus meningitis might not have them in the smear during the first two or three days of the illness. In this child organisms were not found until the fourth week of her illness. That is distinctly unusual so far as the average case is concerned. When they did find influenzal bacilli on smear they

gave serum intraspinally together with human blood serum as complement. Ward and Fothergill,* some years ago, showed that their serum was not bactericidal except in the presence of complement, and there is no use giving it intraspinally without complement because normally, of course, there is no complement in spinal fluid. They evidently were in doubt as to whether she had influenzal meningitis because they continued to give prontylin throughout the hospital stay and in the doses we ordinarily give to children, ¾ to 1 gr. for a pound of body weight. The fact that they obtained no fluid by lumbar puncture on the twentieth day is in favor of influenza-bacillus meningitis, where the spinal cord was surrounded with gelatinous material that acts very effectively in producing block. The spinal fluid findings were consistent with influenza-bacillus meningitis. I should like to know whether any attempt was made to see if the spinal fluid pressure was increased on jugular compression.

Dr. Higgins: My impression is that it was normal.

Dr. Tefft: I do not believe anyone questions the final diagnosis of influenza-bacillus meningitis. She is in the age group in which this kind of meningitis occurs, between six months and two years, and the facts that she had spinal block at the end, and influenzal bacilli were found in the spinal fluid smears make it quite certain that she had at the time of death influenza-bacillus meningitis. I should expect that she would show at postmortem an influenzal meningitis with localized abscesses, more or less walled off, in the sulci especially at the base of the brain, the gelatinous exudate probably covering the spinal cord and producing block.

There are several points that stand out sharply in this story. There were no organisms in the spinal fluid until the tenth day and they were absent in the smears until the fourth week. Then she had marked dilatation of the scalp veins, which is not normal. She also had a tremendous increase in spinal fluid production — or to put it more accurately — a tremendous decrease in the mechanism which produces absorption of the spinal fluid after it has been produced. Those findings point definitely to the fact that she had some sort of process other than typical meningitis, presumably some sort of abscess in the brain substance close to the surface which gave white blood cells in the fluid. Occasionally influenzal bacillus can be demonstrated on culture and not on smear. There was something that was producing a partial venous block, intracranially, something which was interfering very markedly with the absorption of spinal fluid. I think that could best be explained on the basis that she had either an abscess at the base of the brain pressing on the lateral sinus where the lateral sinus goes into the internal jugular vein or a phlebitis or a lateral sinus thrombosis with abscess formation in the sinus and communicating to the brain substance itself. Whether that was unilateral or bilateral, I do not know. I should expect that there were possibly some signs of mastoid involvement, but marked mastoid involvement is not necessary in a child of this age when the bones are thin and the process goes through the bones without definitely involving the mastoid. I think that she had sinus thrombosis with abscess formation communicating with the brain and that she had brain abscess and influenza-bacillus meningitis.

*Ward, H. K., and Fothergill, L. D.: Influenzal meningitis treated with specific antiserum and complement; report of 5 cases. Am. J. Dis. Child. 43:873-881, 1932.

Clinical Discussion

Dr. Higgins: We felt that we were dealing largely with a blood-stream infection rather than a meningeal one. We thought the child had a sinus thrombosis but we placed it in the longitudinal sinus rather than in the lateral sinus. The marked venous return from around the fontanel made us suspect that. How the longitudinal sinus became infected was a problem. We would expect that if it came from influenzal infection of the nasal sinuses it would be of the cavernous sinus instead of the longitudinal sinus. This child did not show signs of cavernous sinus involvement. There was no bulging of the eyes or marked edema of the face. The child had a septic temperature throughout the first four weeks of the disease which is quite in keeping with a blood-stream infection and especially with a sinus thrombosis. It is the same thing we see with streptococcus sinus thrombosis involving the lateral sinus, only here it seems to be the longitudinal sinus. We thought that the infection was largely extradural, that it probably was around the sinus and that the meningitis was characterized by very few cells on that account, but that later the influenzal bacilli did enter the meninges and give a typical influenzal meningitis. I think this case proves very definitely that sulfanilamide was of no value in this influenzal infection because the child received quite a large dose. We attacked the case as one of septicemia rather than one of meningitis.

Clinical Diagnoses

Meningitis, *Bacillus influenzae.*
Septicemia, *Bacillus influenzae.*
Thrombosis, longitudinal sinus.
Hydrocephalus.

Dr. Tefft's Diagnoses

Influenza-bacillus meningitis.
Lateral sinus thrombosis.
Brain abscess.

Anatomical Diagnoses

Influenza-bacillus meningitis.
Thrombophlebitis, superior longitudinal and right lateral sinuses.
Otitis media, acute purulent, bilateral.
Ethmoid sinusitis, acute, purulent, bilateral.
Septicemia, pneumococcus type III.

Pathological Discussion

Dr. Charles S. Kubik: I believe an elevated spinal-fluid pressure will practically always be found in acute meningitis whether there is sinus thrombosis or not.

Autopsy disclosed both meningitis and thrombophlebitis. There was a very large amount of purulent subarachnoid exudate, not only at the base, but also on the convex surfaces, particularly in the upper parietal region. The thrombophlebitis involved the anterior part of the superior longitudinal sinus extending as far back as the anterior fontanel. It was also present in the right lateral sinus, not in the cavernous sinuses. Both middle ears and also the ethmoid cells contained pus. This is the only case of influenzal meningitis in which we have found thrombophlebitis. I think we have had at least one case and possibly others where there was an infected ear. I do not believe we could tell in this case whether it was the infection in the ethmoid or that in the ears which was responsible for the intracranial infection. I suppose it may have been both, the ethmoid being responsible for infection of the longitudinal sinus and the ear for that of the lateral sinus. I should assume that the meningitis was secondary to the thrombophlebitis.

Dr. Wyman Richardson: I should like to ask Dr. Tefft about the positive blood culture of pneumococcus type III.

Dr. Tefft: I do not know what it means. It is a bizarre finding and I cannot see that it makes a great deal of difference except that the child might have had a terminal bronchopneumonia.

Dr. Tracy B. Mallory: We found type III pneumococci in the blood culture at autopsy.

Dr. Higgins: My interpretation was that it was probably secondary infection in the sinuses from type III pneumococci which went in the same way.

Dr. Mallory: We found no significant amount of pneumonia.

The New England
Journal of Medicine

Formerly the
Boston Medical and Surgical Journal

Established in 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

Official Organ of
The Massachusetts Medical Society
The New Hampshire Medical Society
The Vermont State Medical Society

Editorial Board

George G. Smith, M.D. — Dwight O'Hara, M.D.
Joseph Garland, M.D. — John P. Sutherland, M.D.
William B. Breed, M.D. — Stephen Rushmore, M.D.
George R. Minot, M.D. — Hans Zinsser, M.D.
Frank H. Lahey, M.D. — Henry R. Viets, M.D.
Shields Warren, M.D. — Robert M. Green, M.D.
George L. Tobey, Jr., M.D. — Charles C. Lund, M.D.
C. Guy Lane, M.D. — John F. Fulton, M.D.
William A. Rogers, M.D. — A. Warren Stearns, M.D.

Associate Editors

Thomas H. Lanman, M.D. — Donald Munro, M.D.
Henry Jackson, Jr., M.D.

Walter P. Bowers, M.D., Editor Emeritus
Robert N. Nye, M.D., Managing Editor
Clara D. Davies, Assistant Editor

Subscription Terms: $6.00 per year in advance, postage paid, for the United States; Canada, $7.04 per year; $8.52 per year for all foreign countries belonging to the Postal Union.

Material for early publication should be received not later than noon on Saturday.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the New England Journal of Medicine, 8 Fenway, Boston, Mass.

ORGANIZED MEDICINE WILL MEET THE CHALLENGE OF PRESIDENT ROOSEVELT'S COMMITTEE

In recognition of the significant attitude of the national government with respect to the health problems of this country, as set forth in the conference of last July, the American Medical Association called a meeting of its House of Delegates last week to consider plans for the proper procedure to be followed in dealing with the proposals submitted at that conference by representatives of the several bureaus.

The reports of the addresses given by Drs. Abell, Sleyster, Shoulders, and Booth at this meeting indicate a spirit of co-operation between the American Medical Association and the representatives of the government in adjusting the recommendations of the President's committee to methods which will promote adequate medical care of the people but be in harmony with the ideals and traditions of the profession. Such proposals as compulsory health insurance and political control of the practice of medicine will not be endorsed, however.

Any further discussion of what took place at this meeting must wait for accurate reports of the proceedings.

A committee was appointed by the speaker of the House of Delegates to meet with a group of physicians of the federal government to discuss methods of co-ordinating health and welfare activities of private and governmental medical workers.

The names of this committee are: Dr. Irvin Abell, of Louisville, Kentucky, president of the Association, chairman; Dr. Henry A. Luce, of Detroit, Michigan; Dr. Frederic E. Sondern, of New York City; Dr. Walter E. West, of Huntington, West Virginia; Dr. Walter F. Donaldson, of Pittsburgh, Pennsylvania; Dr. Fred W. Rankin of Lexington, Kentucky and Dr. Edward H. Cary, of Dallas, Texas.

DISGUSTINGLY HEALTHY

Still sweet, apparently, are the uses of adversity. For the year 1937, the eighth of the Great Depression, the Bureau of the Census at Washington has announced the lowest infant death rate in the history of the country — a rate of 54.4 deaths for every 1000 live births.

It was, in 1932, as the *New York Times* indicates, a matter of congratulatory comment that, after two years of hard times, the death rate of the nation continued to fall. The authorities warned us, however, to knock on wood, or whatever passes for that ritual in statistical circles; two years of unemployment could not undo a nation's gains in public health. Still, we passed through our first depression and are going into our second, according to the phraseology of the *Times,* and a newborn baby has a third better chance to reach its first birthday than in the normal year of 1925.

This onward surge to better health and lengthened life has not been a result of adverse conditions, much as we might want to point a moral to this effect. We had not reached that stage where

wealth accumulates and men decay, near as we might have been to it in the money-mad twenties. Conditions of life and health have been improving almost steadily for generations, and it happens that our economic distress has not yet been sufficient to affect them unfavorably.

Neither can it be said, despite the *Times'* implication, that because improved health has come under capitalism it has come because of it. "In length of life and in health the masses prospered under the so-called *laissez faire,*" is a statement that smacks of smugness. The fact that the road has been slowly upward under our own system of capitalism does not put a complete stamp of approval on its conduct of the nation's business.

With more enlightenment; with less selfishness under the same or similar or entirely different auspices, our next station may yet be a truly advanced one. With a really bad break in our economic situation; with new fears and high prices and a currency on the rocks, we may find ground lost that will require countless generations to regain.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

SPONTANEOUS RUPTURE OF THE UTERUS FOLLOWING PREVIOUS LOW CERVICAL CESAREAN SECTION

Mrs. A. S., a twenty-four-year-old gravida III, was admitted from the prenatal clinic to the hospital on November 7, 1937, for a repeat cesarean section.

The family history was non-essential. The patient had had no serious illness. Catamenia began at thirteen, were regular every twenty-eight days, and lasted five days without pain. Her last period began February 5, 1937, making her due for delivery November 12. Her first pregnancy resulted in a stillborn infant following a transverse arrest and mid-forceps delivery. The record stated that the baby was delivered with great difficulty. Due to this and her small pelvis her second pregnancy was terminated by a low cervical cesarean section after eight hours of labor with a floating head and no dilatation. The record did not state whether the uterine incision was vertical or transverse. X-ray pelvimetry at this time showed an anteroposterior diameter of 9 cm. and a transverse diameter of 12 cm. Both puerperiums were uneventful. Her present pregnancy had been uneventful except for slight edema of the vulva during the eighth month which was completely relieved by elevation of the foot of the bed for a couple of days. She also stated that for several months she had experienced cramp-like pains for three days at the times when she would have been menstruating had she not been pregnant.

On physical examination the patient was well nourished. The skin and mucous membranes showed no pallor. The heart showed no enlargement; there were no murmurs. The lungs were normal; there were no rales. Her temperature was 98.2°F., pulse 88, and blood pressure 100 systolic, 72 diastolic. The abdomen was rounded, the fundus two fingers below the ensiform. There was no tenderness, and the consistence of the uterus was normal. The vertex was presenting in an LOA position, but it was not engaged. The fetal heart rate was 134, heard in the left lower quadrant.

She was prepared for operation the following morning, but due to a heavy operating room schedule, the operation was postponed until the following day. During that evening she complained of abdominal cramps and backache and had a small amount of bloody show. Immediate operation was then deemed necessary. A low vertical cesarean section was performed within about two hours from the onset of pains. The visceral peritoneum on the lower uterus was incised transversely, and the bladder flap was reflected off easily by blunt dissection. Upon inspection of the exposed uterus, a gap 5 by 6 cm. with the membranes bulging through was seen in the lower uterine segment. The routine ampule of pituitary extract having been administered subcutaneously, a vertical incision was made in the lower segment through the rupture, the membranes were ruptured, and a living female infant was delivered. The baby weighed 6 lb., 11 oz. The operation was finished in the usual manner with two layers of continuous sutures of No. 2 chromic catgut. Sterilization was also performed.

The patient's puerperium was uneventful, and she was discharged on the thirteenth postoperative day with a well-involuted fundus and an incision healed by primary intent.

A series of selected case histories by members of the section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the section.

Comment. This case illustrates two points: first, that a spontaneous rupture of the uterus can occur in a subsequent pregnancy following a low cervical cesarean section; secondly, that in such a case a cesarean section should be promptly performed at the first signs of labor. Had this patient had an active or prolonged labor she would have shown the clinical signs of a ruptured uterus with attendant hemorrhage and shock, and the prognosis would have been quite grave.

GENTLEMEN'S AGREEMENT

The committee representing the physicians, hospitals and insurance companies on the Gentlemen's Agreement is about to hold its first meeting of the season. The physicians on this committee would like to ascertain whether any Massachusetts physicians have had difficulty with their bills after using the forms prepared by the committee.

Some small insurance companies have not yet subscribed to this plan. If any physicians have grievances against insurance companies about bills, the committee will be glad to try to adjust the matter. Please state facts briefly, addressing the undersigned.

HENRY M. LANDESMAN, M.D., *Secretary.*
366 Commonwealth Avenue, Boston.

DEATH

CURRAN — GEORGE L. CURRAN, M.D., of 63 Eagle Street, North Adams, died June 30. He was in his fifty-first year.

He received his degree from the New York University College of Surgeons in 1911. A fellow of the American Medical Association and the American College of Surgeons he was also a member of the Massachusetts Medical Society.

Dr. Curran was surgeon to the North Adams Hospital, a member of the associate staff of St. Luke's Hospital, Pittsfield, and consulting surgeon to the W. B. Plunkett Memorial Hospital, Adams.

MISCELLANY

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR JULY, 1938

DISEASES	JULY 1938	JULY 1937	FIVE-YEAR AVERAGE*
Anterior poliomyelitis	5	30	35
Chickenpox	402	470	375
Diphtheria	8	25	40
Dog bite	1171	1193	1148
Dysentery, bacillary	11	3	2
German measles	34	60	182
Gonorrhea	354	506	557
Lobar pneumonia	147	183	165
Measles	763	672	898
Meningococcus meningitis	3	5	7
Mumps	431	171	254
Paratyphoid A fever	1	0	0
Paratyphoid B fever	1	28	6
Scarlet fever	335	261	297
Syphilis	388	514	394
Tuberculosis, pulmonary	295	392	322
Tuberculosis, other forms	23	34	38
Typhoid fever	5	13	21
Undulant fever	4	3	2
Whooping cough	379	787	637

*Based on figures for the preceding five years.

RARE DISEASES

Anterior poliomyelitis was reported from: Colrain, 1; Fitchburg, 1; Gardner, 1; Haverhill, 1; Watertown, 1; total, 5.

Diphtheria was reported from: Boston, 3; Fall River, 1; Lexington, 1; Salem, 1; Swampscott, 1; Uxbridge, 1; total, 8.

Dysentery, bacillary, was reported from: Boston, 1; Cambridge, 1; Danvers, 3; Fall River, 1; New Bedford, 1; Waltham, 3; Worcester, 1; total, 11.

Encephalitis lethargica was reported from: Lowell, 1.

Malaria was reported from: Malden, 1.

Meningococcus meningitis was reported from: Ashfield, 1; Chicopee, 1; Quincy, 1; total, 3.

Paratyphoid A fever was reported from: New Bedford, 1.

Paratyphoid B fever was reported from: Springfield, 1.

Pfeiffer-bacillus meningitis was reported from: Brookline, 1.

Pellagra was reported from: Bridgewater, 1; Marlboro, 1; Waltham, 1; total, 3.

Septic sore throat was reported from: Belmont, 2; Boston, 4; Colrain, 1; Fall River, 2; North Westport, 1; Pittsfield, 1; total, 11.

Tetanus was reported from: Cambridge, 1; Fairhaven, 1; Somerville, 1; total, 3.

Trachoma was reported from: Boston, 2; Lynn, 1; total, 3.

Trichinosis was reported from: Springfield, 1; Westwood, 1; total, 2.

Typhoid fever was reported from: Abington, 1; Everett, 1; Lawrence, 1; Longmeadow, 1; Pittsfield, 1; Shrewsbury, 1; total, 6.

Undulant fever was reported from: Beverly, 1; Orange, 1; Whitinsville, 2; total, 4.

Diphtheria was reported at a record low figure for the third consecutive month.

Chickenpox, mumps and scarlet fever were reported above the five-year average. Tuberculosis (other forms) showed record low incidence.

Lobar pneumonia, pulmonary tuberculosis, meningococcus meningitis and whooping cough were reported below the five-year average.

Anterior poliomyelitis continued to show low incidence.

Except for 1936, the figure of which was equalled, undulant fever showed record high incidence.

German measles and measles were reported below the five-year average.

Typhoid fever showed record low incidence for the third consecutive month.

The reported incidence of dog bite continued at a high figure. Several cases of animal rabies were reported. Previously reported foci in Reading and Worcester were active. New foci were noted in Attleboro and Milton.

CORRESPONDENCE

THE RELATION OF HUMAN ENCEPHALITIS TO EQUINE DISEASE

To the Editor: The note published this week by Dr. Fothergill and his associates of Harvard Medical School stating that a case of encephalitis in a child has been definitely proved to be due to the Eastern strain of equine encephalomyelitis virus has been confirmed in three additional cases by Dr. Leslie T. Webster of the Rockefeller Institute of New York. This immediately raises the question as to the importance the problem may assume in this

state. Since the equine disease is already fast declining, as it is known to do with the approach of cold weather, there does not seem to be any prospect of an immediate increase in human cases. The problem to be faced is largely one of the future.

This department has under investigation several similar cases in children which resemble that reported by Dr. Fothergill. Clinically and pathologically these cases appear to belong to a different type of encephalitis from those previously recognized in this state. So far the suspect cases have been limited to the area in which the equine disease is prevalent, namely, southeastern Massachusetts.

To date no multiple cases in families have been reported and the department has no evidence as to how the infection is conveyed. In view of our knowledge that the disease in horses is transmitted by mosquitoes, it is reasonable to assume that it is conveyed to man in the same way. Accordingly it is suggested that children in the area affected be protected from mosquitoes by screening, by sprays and by keeping them indoors especially after dusk.

The department is very anxious to know of all cases of illness in children in which encephalitis is even suspected. All the resources of the State are being mobilized to gather information which may have a bearing on the distribution and future importance of the disease in man.

The Massachusetts Division of Livestock Disease Control is planning to push the immunization of horses with the hope of having every horse in the state immunized by June 1, 1939. If this is done, it may be that the equine disease can be controlled, thereby eliminating that probable source of infection.

ALTON S. POPE, M.D., *Deputy Commissioner,*
Massachusetts Department of Public Health.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

To the Editor: In addition to the articles enumerated in our letter of August 15 the following have been accepted:

Abbott Laboratories

Sulfanilamide — Abbott, Ampoules (crystals) 1 gm.

Gane & Ingram, Inc.

Ephedrine Alkaloid Anhydrous
Ephedrine Alkaloid Hemihydrate

Gilliland Laboratories

Typhoid Vaccine (Gilliland), 50 cc. size package

McKesson & Robbins, Inc.

McKesson's Cod Liver Oil Concentrate in Oil

Upjohn Company

Tablets Phenobarbital — Upjohn, ¼ gr.
Tablets Phenobarbital — Upjohn, ½ gr.
Tablets Phenobarbital — Upjohn, 1½ gr.
Staphylococcus Mixed Vaccine
Typhoid Vaccine
Typhoid Paratyphoid Mixed Vaccine

PAUL NICHOLAS LEECH, *Secretary.*

535 North Dearborn Street,
Chicago, Illinois.

REPORT OF MEETING

EASTERN HAMPDEN MEDICAL ASSOCIATION

The opening meeting of the fall session of the Eastern Hampden Medical Association was held on September 15 at the Oaks Hotel in Springfield.

The secretary regretfully reported the death of Dr. W. Edward Gorman, of Springfield, on September 9, whereupon the President appointed a committee consisting of Drs. R. E. Dickson, of Holyoke, and Erdix Smith, of Springfield, to draw up an appropriate resolution, Dr. Gorman having been a member of this society for many years.

Dr. James J. Grace, of Springfield, presented a case of hernia of an intervertebral disk which was erroneously treated for more than a year as arthritis of the spine. Complete recovery followed laminectomy at the Lahey Clinic.

Dr. M. J. Kranichuck, of South Hadley Falls, then presented a case of bilateral tubal pregnancy in a thirty-two-year-old multipara with rupture of the left tube into the cul-de-sac, and with recovery following early laparotomy. Only seven similar cases had been reported in the literature.

Dr. Erdix Smith, previously of Springfield, discussed the case of a forty-two-year-old machinist who slipped on a ramp, sustained a fracture of the body of the second lumbar vertebra and subsequently developed marked anesthesia extending up to the neck and shoulders. It was felt that the symptoms were due to a compression hemorrhage, but that, inasmuch as the patient refused spinal puncture, the causal factor was still in doubt.

The scientific session was followed by a turkey dinner served to the members.

The next meeting will be held October 6 at The Oaks Hotel.

J. JOSEPH KLAR, *Secretary.*

NOTICES

LAWRENCE CANCER CLINIC

The regular Lawrence Cancer Clinic, to be held at the Lawrence General Hospital, 1 Garden Street, Lawrence, on Tuesday, October 4, at 10:00 a. m., will be a demonstration and teaching clinic for physicians, with Dr. Channing C. Simmons, of Boston, associate in surgery in the graduate courses in medicine at Harvard Medical School, surgeon-in-chief to the Collis P. Huntington Memorial Hospital, member of the Cancer Commission of Harvard University, and visiting surgeon to the Massachusetts General Hospital, present as consultant. Physicians of the north half of Essex County are invited to accompany any of their patients whom they desire to have this service or to send them with a note. A report will be returned to every physician who sends a patient. The service is gratis. Any physician is welcome to attend the clinic.

This clinic is endorsed by the Committee on Postgraduate Instruction of the Massachusetts Medical Society.

ROY V. BAKETEL, M.D.,
CHARLES J. BURGESS, M.D.,
JOHN J. MCARDLE, M.D.,
HARRY H. NEVERS, M.D.,
THOMAS V. UNIAC, M.D.,
J. FORREST BURNHAM, M.D., *Chairman.*

BOSTON PSYCHOANALYTIC INSTITUTE

The Boston Psychoanalytic Institute announces three Sigmund Freud Fellowships in Psychoanalytic Training, beginning September, 1939. The fellowships are open to graduates of Class A medical schools who have had at least one year of general hospital training and two years' work in psychiatry. Applications must be in before February 1, 1939.

For further information, write to Dr. M. Ralph Kaufman, chairman of the Educational Committee, 82 Marlboro Street, Boston.

CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS, UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

Clinic	Date	Orthopedic Consultant
Salem	October 3	Harold C. Bean
Haverhill	October 5	Arthur T. Legg
Lowell	October 7	Albert H. Brewster
Gardner	October 11	Mark H. Rogers
Brockton	October 13	George W. Van Gorder
Pittsfield	October 17	Francis A. Slowick
Springfield	October 19	Garry deN. Hough, Jr.
Worcester	October 21	John W. O'Meara
Fall River	October 24	Eugene A. McCarthy
Hyannis	October 25	Paul L. Norton

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY, INC.

The next examinations (written and review of case histories) for Group B candidates will be held in various cities of the United States and Canada on Saturday, November 5, at 2:00 p. m., and on Saturday, February 4, 1939. Application for admission to the written examination scheduled for February 4 must be filed on an official application form in the office of the secretary at least sixty days prior to this date (or before December 4).

The general oral, clinical and pathological examinations for all candidates (Groups A and B) will be conducted by the entire board, meeting in St. Louis, Missouri, on May 15 and 16, 1939. Application for admission to Group A examinations must be on file in the secretary's office before April 1, 1939.

For further information and application blanks, address Dr. Paul Titus, secretary, 1015 Highland Building, Pittsburgh (6), Pennsylvania.

INTER-STATE POSTGRADUATE MEDICAL ASSOCIATION

The twenty-third International Assembly of the Inter-State Postgraduate Medical Association of North America will be held in the public auditorium of Philadelphia, Pennsylvania, October 31, November 1, 2, 3 and 4. All scientific and clinical sessions will take place in the auditorium. Hotel headquarters will be the Benjamin Franklin Hotel.

The members of the medical profession of Philadelphia are correlating for the clinics an abundance of hospital material representing various types of pathologic conditions which will be discussed by the contributors to the program.

In the neighborhood of eighty distinguished teachers and clinicians will appear on the program, and a tentative list of speakers may be found on page vii of the advertising section of the *Journal*. The subjects and speakers have been selected to consider practically all the topics of greatest interest to the medical profession in general.

A full program of scientific and clinical sessions will take place every day and evening of the assembly, starting each morning at 8:00 o'clock. On account of the fullness of the program, restaurant service will be available at the auditorium at moderate prices.

The members of the profession are urged to bring their wives with them as a very excellent program is being arranged for their benefit by the ladies' committee. Philadelphia has many places of historical and other interests, which will make this year's program especially attractive to them.

Pre-assembly and post-assembly clinics will be held in the Philadelphia hospitals on Saturday, October 29, and Saturday, November 5.

It is very important that you make your hotel reservations early, by writing Mr. Thomas E. Willis, chairman, Hotel Committee, Chamber of Commerce Building, 12th and Walnut Streets, Philadelphia, Pennsylvania.

The association, through its officers and members of the Program Committee, extends a very hearty invitation to all members of the profession in good standing in their state and provincial societies to attend the assembly. The registration fee is $5.00.

Dr. Elliott P. Joslin, *President*,
Boston, Mass.,

Dr. George W. Crile, *Chairman*, Program Committee,
Cleveland, Ohio,

Dr William B. Peck, *Managing-Director*,
Freeport, Illinois.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held on Tuesday, September 27, in the Peter Bent Brigham Hospital amphitheater (Shattuck Street entrance), at 8:15 p. m.

PROGRAM

Presentation of cases.

The Diagnosis of Fevers in Practice. Dr. Philip Manson-Bahr, London, England.

Dr. Richard P. Strong will preside. Medical students and physicians are cordially invited to attend.

Robert Zollinger, M.D., *Secretary*.

REMOVAL

Salvatore Sannella, M.D., announces the removal of his office to 83 Maple Street, Springfield, Massachusetts.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Monday, September 26

Tuesday, September 27

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

*8:15 p. m. Harvard Medical Society. Peter Bent Brigham Hospital amphitheater (Shattuck Street entrance).

Wednesday, September 28

*12 m. Clinicopathological conference. Children's Hospital amphitheater.

Thursday, September 29

*10:30 a. m. Psychiatric Rounds. Boston City Hospital. Rounds will start at Room 916 on the ninth floor of the Medical Building.

Friday, September 30

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

Saturday, October 1

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

September 26 and 27 — Symposium on Occupational Disease. Page 145, issue of July 28.

September 27 — Harvard Medical Society. Notice above.

October 3-15 — Inactive Status Training Course. Page 288, issue of August 25.

October 4 — Lawrence Cancer Clinic. Page 450.

October 4 and 5 — Massachusetts Medico-Legal Society. Page 365, issue of September 8.

October 7 and 8 — Conference on Rural Medicine. Page 180, issue of August 4, and page 407, issue of September 15.

October 8 and November 15 — American Board of Ophthalmology. Page 282, issue of February 10.

OCTOBER 13 — Pentucket Association of Physicians. 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

OCTOBER 17, 1938 and FEBRUARY 20, 1939 — American Board of Internal Medicine, Inc. Page 216, issue of August 11.

OCTOBER 17-21 — Clinical Congress of the American College of Surgeons, New York City.

OCTOBER 24-26 — Academy of Physical Medicine, Scientific Session. Washington, D. C.

OCTOBER 24 - NOVEMBER 4 — Graduate Fortnight of the New York Academy of Medicine. Page 181, issue of August 4.

OCTOBER 25-28 — American Public Health Association. Page 321, issue of September 1.

OCTOBER 31, NOVEMBER 1, 2, 3 and 4 — Inter-State Postgraduate Medical Association. Page 451.

NOVEMBER 5 — American Board of Obstetrics and Gynecology. Page 145, issue of July 28.

NOVEMBER 5, 1938; FEBRUARY 4, MAY 15 and 16, 1939 — American Board of Obstetrics and Gynecology. Page 451.

MARCH 27-31, 1939 — American College of Physicians. Page 36, issue of July 7.

SEPTEMBER, 1939 — Boston Psychoanalytic Institute. Page 450.

BOOKS RECEIVED FOR REVIEW

Cancer: With special reference to cancer of the breast. R. J. Bahan. 844 pp. St. Louis: The C. V. Mosby Company, 1938. $10.00.

British Periodicals of Medicine: A chronological list. W. R. Lefanu. 93 pp. Baltimore: The Johns Hopkins Press, 1938. $1.25.

The Troubled Mind: A study of nervous and mental illnesses. C. S. Bluemel. 520 pp. Baltimore: The Williams & Wilkins Company, 1938. $3.50.

Papers on Psycho-Analysis. Ernest Jones. Fourth edition. 643 pp. Baltimore: William Wood & Company, 1938. $8.00.

The Principles and Practice of Medicine: Designed for the use of practitioners and students of medicine. Revised by Henry A. Christian. Thirteenth edition. 1424 pp. New York and London: D. Appleton-Century Company, 1938. $9.00.

The Pathology of Diabetes Mellitus. Shields Warren. Second edition. 246 pp. Philadelphia: Lea & Febiger, 1938. $4.75.

Babies Are Human Beings. C. Anderson Aldrich and Mary M. Aldrich. 128 pp. New York: The Macmillan Company, 1938. $1.75.

Triumph Over Pain. René Fülöp-Miller. 438 pp. Indianapolis and New York: The Bobbs-Merrill Company, 1938. $3.50.

Leaves from a Surgeon's Case-Book. James Harpole. 300 pp. New York: Frederick A. Stokes Company, 1938. $2.75.

Dysmenorrhoea. Albert A. Davis, 254 pp. London, New York and Toronto: Oxford University Press, 1938. $4.50.

Refraction of the Eye. Alfred Cowan. 319 pp. Philadelphia: Lea & Febiger, 1938. $4.75.

Guiding Human Misfits. Alexandra Adler. 88 pp. New York: The Macmillan Company, 1938. $1.75.

Heart Disease and Pregnancy. Crighton Bramwell and Edith A. Longson. 194 pp. London, New York and Toronto: Oxford University Press, 1938. $3.00.

A Textbook of Nursing Practice. Hester K. Frederick and Ethel Northam. Second edition (revised). 418 pp. New York: The Macmillan Company, 1938. $3.00.

Aids to Histology. Alexander Goodall. Fourth edition. 151 pp. Baltimore: William Wood & Company, 1938. $1.25.

Anus, Rectum, Sigmoid Colon: Diagnosis and treatment. Harry E. Bacon. 855 pp. Philadelphia, Montreal and London: J. B. Lippincott Company, 1938. $8.50.

Subacute and Chronic Pericardial and Myocardial Lesions Due to Non-Penetrating Traumatic Injuries. Erik Warburg. 147 pp. New York and London: Oxford University Press, 1938. $6.00.

Proceedings of the Fourth Conference on Psychiatric Education. 345 pp. New York: The National Committee for Mental Hygiene, Inc., 1938. $1.00.

The Technique of Contraception. Eric M. Matsner and Frederick C. Holden. 50 pp. Baltimore: The Williams & Wilkins Company, 1938.

The History of Bacteriology. William Bulloch. 422 pp. London, New York and Toronto: Oxford University Press, 1938. $3.75.

You Can Sleep Well: The A B C's of restful sleep for the average person. Edmund Jacobson. 269 pp. New York and London: Whittlesey House, McGraw-Hill Book Company, Inc., 1938. $2.00.

William Alanson White: The autobiography of a purpose. William A. White. 293 pp. New York: Doubleday, Doran & Company, Inc., 1938. $3.00.

A College Textbook of Pharmaceutical Botany. Heber W. Youngken. Sixth edition. 793 pp. Philadelphia: P. Blakiston's Son & Company, Inc., 1938.

Handbook of Histological and Cytological Technique. R. R. Bensley and S. H. Bensley. 167 pp. Chicago: The University of Chicago Press, 1938. $2.00.

Surgical Pathology. William Boyd. Fourth edition. 886 pp. Philadelphia and London: W. B. Saunders Company, 1938. $10.00.

The Principles and Practice of Obstetrics. Joseph B. DeLee. Seventh edition. 1211 pp. Philadelphia and London: W. B. Saunders Company, 1938. $12.00.

Experience in the Management of Fractures and Dislocations: Based on an analysis of 4390 cases. Edited by Philip D. Wilson. 1036 pp. Philadelphia: J. B. Lippincott Company, 1938. $15.00.

The New International Clinics: Original contributions — clinics; and evaluated reviews of current advances in the medical arts. Edited by George M. Piersol. Volume 3, N. S. I. 341 pp. Philadelphia, Montreal and New York: J. B. Lippincott Company, 1938. $3.00.

Feminine Hygiene in Marriage. A. F. Niemoeller. 155 pp. New York: Harvest House, 1938. $2.00.

Laboratory Manual of Hematologic Technic: Including interpretations. Regena Cook Beck. 389 pp. Philadelphia and London: W. B. Saunders Company, 1938. $4.00.

Chronic Rheumatic Diseases: Being the fourth annual report of the British Committee on Chronic Rheumatic Diseases appointed by the Royal College of Physicians. C. W. Buckley. 160 pp. New York: The Macmillan Company, 1938. $3.25.

The Spectacle of a Man. John Coignard. 252 pp. New York: Jefferson House, Inc., 1937. $2.50.

BOOK REVIEWS

Treatment of Some Chronic and "Incurable" Diseases. A. T. Todd. 203 pp. Baltimore: William Wood & Company, 1937. $3.00.

This book is the presentation of an English physician's observations on the treatment of chronic diseases. Todd is obviously an individualist who questions the whys, hows and whens, rather than accepts unquestioningly "the traditional therapeutics which have years of inadequacy to support them." Apparently he has studied his individual patients more fully than he has the agents of disease. As a result his ideas of treatment often differ from the standard, accepted, routine methods found in most textbooks of medicine.

The chapters deal with diabetes mellitus, epilepsy, false

phthisis, asthma and hay fever, postencephalitic parkinsonism, Graves' disease and toxic goiter, parenchymatous nephritis, duodenitis and hepatic defect, false heart disease, constipation, acute rheumatism and chorea, ulcerative colitis, chronic rheumatism and cancer. The last subject is dealt with in three chapters — one "Theoretical," one "Therapeutic" and the third "Results and Conclusions." The appendixes are divided into four chapters giving practical details of laboratory methods, diets, prescriptions, and formulas and intravenous medication.

The book is full of interesting reminders of old-time medications which Todd finds have still a place in present-day therapeutics. Perhaps one of the most striking is his use of pectin in the treatment of colitis and chronic diarrheas (page 192). In 1925, Francis Palfrey in his *The Art of Medical Treatment* suggested a similar treatment in the use of large quantities of ripe pears for chronic diarrhea. This instance is typical of many others. In other words, while other authors have made isolated comparisons with former methods of treatment, no one before Todd appears to have observed and made a study of so many in so many different diseases.

The themes of the book are the individual and the outlet in the form of disease which that individual has taken. Curiously enough, the words psyche, psychical or psychiatric are not stressed in the text, although the author's approach to his patient, his emphasis on the fact that "there can be no disease of one system except all the other systems be also affected more or less," makes one feel that his *is* a psychological approach, in fact if not in word. The reviewer believes value would have been added to an excellent clinical treatise had this "system" been more consciously taken into account.

However, the book is well worth while. It will undoubtedly appeal to the man who has tried accepted routine measures with unsatisfactory results. It is written by a man who has been in general practice for twenty years but who is no longer satisfied with routine procedure. His observations are not snap judgments; they are based on long personal experience. His case presentations show a recognition of the importance of personal follow-up of each patient. This fact in itself adds definite value to the book and explains statements which offhand appear rather bizarre to the man who works only in the laboratory or to the one who has not given time to study the individual who came to him for help.

Diseases of Women for the General Practitioner. Paul Titus. Edited by Morris Fishbein. 320 pp. New York: National Medical Book Company, Inc., 1937. $4.00.

This is a new book designed for the use of the general practitioner and thus chiefly emphasizes diagnosis. Treatment, except major surgery which is regarded as outside the province of those for whom the book is written, "is discussed as practically as possible," with a reasonable degree of completeness for a small book.

The procedure adopted by the author attempts to combine two somewhat irreconcilable points of view. One is stated in the preface: "how gynecologic problems present themselves to the general practitioner"; the other is set forth in the table of contents which has the usual classification of the diseases in gynecology, namely, by organs, noting the effects of trauma, infection and tumors. For example, the problem of cancer of the body of the uterus does not present itself as such, although it assumes that aspect when the diagnosis has been made. It may present itself under the disguise of an irritating leukorrhea appearing after the menopause.

The defects in arrangement are really serious. For example, if the patient's chief complaint is metrorrhagia, one looks in the index and finds one reference to page 273, where there are thirteen lines devoted to this important symptom. As to cause: "The etiology is about the same as that of menorrhagia." The index contains one reference to menorrhagia: "Etiology. The causes of menorrhagia have been mentioned repeatedly and have already been described; they need only be summarized here." Then follow twelve lines in which syphilis is mentioned as an outstanding cause of menorrhagia. Apparently one takes syphilis for granted for it is not mentioned in the table of contents nor in the index.

However, when the obstacles of arrangement and defective index have been surmounted, the book should prove useful as it specifies many worth-while details in treatment. Success in office practice in gynecology depends in no small part on meticulous attention to details.

The Management of Fractures, Dislocations, and Sprains. John A. Key and H. Earle Conwell. Second edition. 1246 pp. St. Louis: The C. V. Mosby Company, 1937. $12.50.

The second edition of Key and Conwell's textbook is a large single volume treatise. The first part consists of ten chapters on the principles of the subject, including first-aid, complications, compound fractures, workmen's compensation laws and medicolegal aspects. The second part consists of seventeen chapters and is devoted to the diagnosis and treatment of specific injuries. In each chapter are given the surgical anatomy, including that of the bones, joints and soft parts, the method of examination, the type of fracture, the mechanism of the fracture, the diagnosis and the treatment. Under treatment are presented closed manipulation, immobilization, traction, operative methods of reduction and after-care.

A special chapter on "Fractures of the Skull and Brain Trauma" has been prepared by the late Charles E. Dowman, and another on "Fractures of the Jaw and Related Bones of the Face" by James B. Brown. The chapters on fractures of the spine and hip have been extensively rewritten.

One hundred pages are devoted to injuries in the region of the hip, and forty-two to fractures of the neck of the femur. Fractures at the base of the femoral neck are classified as trochanteric. It is pleasing to see the diagnostic mensurations of Roser-Nelaton, Bryant and Morris well described and illustrated. The Whitman, Leadbetter, and Cotton closed methods are described under operative procedures. With internal fixation are given Smith-Petersen's direct-vision nailing, O'Meara's modification and several procedures using mechanical guides (Wedcott, Johnssen, and so forth). The technic of Austin Moore is described, and reference is made to the methods of Telton and Ransohoff, Gaenslen and Knowles, and Henderson.

Another chapter, also of one hundred pages, is devoted to fractures in the region of the elbow. The low transverse fracture of Kocher is erroneously called dicondylar, instead of diacondylar. (The same error is made in a standard textbook on fractures published within a few years.) The term dicondylar is of course synonymous with intercondylar. These diacondylar fractures and separations of the lower humeral epiphysis are confused. The recitation of specific tests for the integrity of the ulnar, radial and median nerves would have been desirable. Recording progress in after-care by tracings or goniometer readings should have been mentioned. Under Volkmann's contracture, which is excellently presented, the reference

should be to S. G. (not S. J.) Jones. An emphatic distinction should be made between separations of the capitellar epiphysis and fractures of the capitellum. Insistence on the operative reposition of badly displaced epiphyses of the radial head should have been made. The general excellence of the chapter so impressed the reviewer that these few errors of commission and omission came to mind.

As an introductory chapter that on pathologic fractures is probably adequate, but it seems rather cursory in comparison with the detailed exposition of the others.

The work should be on the shelves of every doctor who undertakes the treatment of fractures, be he a general practitioner or an operating surgeon. The presentation is clear and thoroughly practical. Although all good methods of treatment are not presented, those which are given are sound and, in case of the commoner lesions, obviously supported by considerable personal experience. It is a well-made volume, including a double-column index of twenty-four pages. The illustrations, over twelve hundred in number, are well chosen and show to advantage on the highly calendered paper. The publishers are to be congratulated on the physical qualities of the book.

Milestones in Medicine: Laity lectures of the New York Academy of Medicine. 276 pp. New York and London: D. Appleton-Century Company, 1938. $2.00.

The New York Academy of Medicine in inaugurating and publishing these lectures has performed a valuable service and one of which it is strongly to be hoped the public will take advantage. For the lecturers not only set forth with unimpeachable accuracy certain facts and ideologies of medicine but through the employment of the historical method show how medical knowledge has been evolved. Thus is furnished not merely understanding but the most fascinating sort of reading. There are seven lectures in the current series: "The Historical Background of Psychiatry" by Smith E. Jelliffe; "The Mechanisms of Heredity" by Charles A. Stockard; "Medicine at Sea in the Days of Sail" by Kurt Vogel; "The Evolution of the Human Brain" by Frederick Tilney; "The History of Medical History" by Henry Sigerist; "The History of Leprosy" by Newton E. Wayson; and "The Story of the Glands of Internal Secretion" by Walter Timme.

Intended though they are for laymen there is much in these lectures which from the historical standpoint will come as new to the great majority of physicians. They can read the lectures with pleasure and profit as well as recommend them to their patients.

Surface and Radiological Anatomy: For students and general practitioners. Arthur B. Appleton; William J. Hamilton, and Ivan C. C. Tchaperoff. 311 pp. Baltimore: William Wood & Company, 1938. $5.50.

From time to time books have appeared on surface anatomy and on radiology and in each instance the authors have hewed straight to the point. This book is an attempt to bridge the gap between these separate disciplines by the simple process of combining them. The effort merits serious consideration on the part of those whose responsibility it is to teach students in clinical surgery and in radiology. The emphasis of the authors on morphology is well placed and will surely aid students in carrying over what they learn in anatomy during the first and second years into the third and fourth years.

There are three hundred and thirty-eight illustrations, which are clear and should result in much profit to practitioners and students. Of necessity, the descriptions are brief, but provocative of thought. The nomenclature is that of the Birmingham Revision of 1933, as adopted by the Anatomical Society of Great Britain and Ireland.

Vitamin B_1 (Thiamin) and Its Use in Medicine. Robert R. Williams and Tom D. Spies. 411 pp. New York: The Macmillan Company, 1938. $5.00.

Vitamin B_1 (thiamin), so recently made available for clinical use in its pure form, is proving to be of great value in the treatment not only of beriberi but also of polyneuritis associated with chronic alcoholism, pellagra, pregnancy and dietary deficiency and of peripheral neuritis with edema of undetermined origin. This book brings to the physician all that is known regarding this remarkable substance, with the clinical data as well as the history and the experimental evidence from the laboratory. It is a timely and remarkably complete publication and should prove of inestimable value to all thoughtful physicians. There are numerous charts and tables, an extensive bibliography and an index.

Aids to Embryology. Richard H. Hunter. Third edition. 178 pp. Baltimore: William Wood & Company, 1938. $1.25.

The first edition of this little book appeared ten years ago. In its third edition, it is freshened by the inclusion of a new chapter on genetics with other chapters rewritten to bring the material up to date. Attention is frequently called to those anatomical facts which explain the significance of the common abnormalities found in the dissecting room and in the obstetric wards. The book will appeal as a quick means of review to students preparing for examinations.

Major Endocrine Disorders. S. Levy Simpson. 184 pp. London: John Bale Medical Publications, Ltd., 1938. 10s. 6d.

Advances in endocrinology, as the foreword to this book truly states, have increased so rapidly that it is difficult for the busy practitioner to keep pace with them. Encyclopedic works of reference there are, but on too large a scale for his needs, and treatises on the physiology of the ductless glands, though relatively numerous, rarely include the therapeutic aspects. This is the state of affairs which the author undertakes to remedy, and, in the opinion of the reviewer, he has made a surprisingly good job of it. For though the book is small enough to slip easily into the pocket he has managed to cover pretty much the whole ground, including all the important recent advances. On debatable points he very fairly states the evidence pro and con and then goes on to give his own opinion, based on a very considerable experience. He has even found space for essential bits of history. The book is well printed and the numerous illustrations, mostly from photographs, are all pertinent and instructive. It will be a worthwhile addition to the library of any general practitioner, intern or medical student. The man who is reviewing for examinations will doubtless bless it.

Studies from the Rockefeller Institute for Medical Research. Volumes 106 and 107. 634 pp. and 603 pp. New York: The Rockefeller Institute for Medical Research, 1938. $2.00 each.

Volume 106. This volume includes the usual wide range of subjects. From the clinical standpoint, the article by Goodner and co-workers on type-specific antipneumococcic rabbit serum is of especial importance. In addition the continued studies of Rhoads on sprue and pernicious anemia are of definite interest. Northrop's article on concentration and purification of bacteriophage is a most valuable summarization of the nature of the staphylococcic bacteriophage; it leads to the conclusion that the formation of phage is probably analogous with an autocatalytic protein enzyme system.

Volume 107. Of special interest from the clinical standpoint are the continued reports of Goodner and Horsfall on antipneumococcic rabbit serum, the study of Swift on the action of sulfanilamide in rheumatic fever and that of Lewis on the changes in the metabolic rate and cardiac output with age.

Essentials of Obstetrical and Gynecological Pathology: With clinical correlation. Marion Douglass and Robert L. Faulkner. 187 pp. St. Louis: The C. V. Mosby Company, 1938. $4.75.

The text presents in clear concise form the gross and microscopic pathology of the female generative organs and has a definite place in the library of the gynecologist for as the text implies anyone pretending to specialize in gynecology must know the related pathology.

Once read it becomes an excellent reference book and one which should be on every gynecologist's bookshelf and available to every student of pathology. The handling of each item from the surgical point of view makes the book doubly valuable for it describes the indicated modern treatment for each disease.

One wonders why no mention is made of the diseases of the mammary gland — certainly an organ, with the exception of the rare male tumor, peculiar to the gynecological and obstetric patient.

Massage and Remedial Exercises in Medical and Surgical Conditions. Noël M. Tidy. Third edition. 456 pp. Baltimore: William Wood & Company, 1937. $5.25.

The subject of fractures is probably the most important one in a work of this kind. Accordingly, it is discussed in the first forty-eight pages. The author systematically describes the varieties of fractures; their etiology and symptomatology; the complications; the process of repair; supportive and physical therapeutic treatment. Under the latter, reflex stroking, effleurage, kneading and friction are taken up as different aspects of massage. The reason for each of these forms is clearly stated. Similar treatment is accorded to "relaxed passive movement" and "assistive active movement," when they are discussed under the general title of "movements." Splinting and bandaging are not only carefully treated in the text but also illustrated by means of excellent diagrams and photographs. Students will greatly appreciate this valuable method of teaching.

The neurologic conditions are considered from the anatomical and pathological points of view. Exercises with pulleys and suspension apparatus are depicted graphically. This is rather a rare treat, for as a rule, such drawings do not appear in a work of this sort.

To the subject of peripheral nerve lesions are allotted about twenty pages. The reviewer believes that the author should have been more generous with space. A similar disproportion between the number of pages given to central- and peripheral-nervous-system lesions is found in many textbooks on neurology, but from the standpoint of physical therapy, the conditions affecting the peripheral nervous system deserve more attention.

Some deformities call for surgical operations, and others for braces, corsets and belts; but practically all of them, at one stage or another, derive benefit from massage and particularly from carefully chosen exercises. The author was cognizant of the importance of the subject and devoted seventy-five pages to its discussion. This portion of the book is a veritable treatise on orthopedics. The various conditions, — congenital and acquired, structural and functional, — the diagnosis of each condition based on signs and symptoms, and the detailed methods of treatment in accordance with each stage of the disease, are all so graphically presented that the reader should have no difficulty in recognizing these conditions in actual practice and in properly administering massage and remedial exercises.

In subsequent editions it might be well for the publisher to use somewhat larger print.

Practical Clinical Gynecology. Henry C. Falk. 393 pp. New York: American Journal of Surgery, Inc., 1938. $5.00.

This short but detailed volume is practical so far as it goes, but it omits many practical details in favor of rather lengthy and not particularly practical discussions concerning interesting gross and microscopical studies of various gynecological diseases. In the first chapter on examination of the patient, details as to what to do when examining a virgin or a young woman are left unsaid, as is any precaution to be taken when examining a normal or abnormal pregnant woman. At least a word of warning should have been included regarding the danger of examining those with bizarre obstetrical problems without proper surgical preparation.

The second chapter contains a minute description of preoperative and postoperative routine, stressing mainly the author's previous writings in this field, and while good, the topic hardly deserves so much space. The section on the pathology of abortion is excellent, as well as those on the classification and suggested treatment. Gonorrhea is well handled except for devoting too much space to operative considerations.

The fifth chapter repeats much of the second while discussing the familiar "fibroid," and one shudders at the dangerous technic that is advised for tying the uterine vessels in doing a routine hysterectomy.

The last half of the book takes up the ovaries, endometriosis, carcinoma, ectopic pregnancy and the relaxed pelvic floor in brief but well-chosen style, but the final chapter on office work is woefully sketchy. The section on sterility is well done and will introduce many to the Sovak reamer.

The text is easy to read, makes one think and unquestionably brings to mind many helpful gynecological tricks of diagnosis and treatment.

Fevers for Nurses. Gerald E. Breen. 199 pp. Baltimore: William Wood & Company, 1938. $2.00.

This is a series of lectures delivered to nurses in the London County Council's infectious hospitals to prepare them for a certificate in fever nursing. One cannot see how this book would be of any interest to American physicians. It is a simple syllabus for local use in English hospitals.

The Single Woman and Her Emotional Problems. Laura Hutton. Second edition. 173 pp. Baltimore: William Wood & Company, 1937. $1.50.

No book can settle the specific problems of particular individuals; what Dr. Hutton tries to do is to orient the single woman psychologically with regard to her problems so that she may hope to achieve her own solutions. There is evidence that she has in considerable measure succeeded; this is the second edition in English, and the book has been translated into Dutch and Norwegian. At any rate Dr. Hutton's psychology seems sound, and she exhibits a broad and tolerant understanding: it is a safe book to recommend.

Physiology of the Nervous System. J. F. Fulton. 675 pp. London, New York, Toronto: Oxford University Press, 1938. $6.00.

In the thirty-odd years since Sherrington's *The Integrative Action of the Nervous System,* published in 1906, there has been a constant flow of new material from many laboratories elucidating the complex action of the brain, spinal cord and peripheral nerves. Some of the advances were published in book form by Fulton in 1926 in his *Muscular Contraction and the Reflex Control of Movement,* but the latest material, still in various journals, has been widely scattered until the issue of the book under review. In this volume, handsomely printed with exceptional, new, line drawings as illustrations, Fulton has brought the subject up to date with complete references to the literature. He seems to have missed no recent work of importance, and moreover, each chapter is headed by a brief historical introduction. This is an outstanding and fundamental text, indispensable to all neurologists, both experimental and clinical. It is a worthy contribution, reflecting the steps in a rapidly growing subject and will stand with Sherrington's book, mentioned above, as a classic of neurology.

Cause and Prevention of Disease. William H. Perkins. 713 pp. Philadelphia: Lea & Febiger, 1938. $7.50.

In this volume the reader will find an almost encyclopedic treatise on the causes of disease generally recognized by authorities and the protective and preventive measures employed in dealing with illness and certain kinds of injuries. As a basis for the subjects discussed the author gives a comprehensive definition of health with explanations of the factors which promote or interfere with the normal functions of the human body. In this is included the role of heredity and the environmental influences of early life. Later chapters take up practically almost all diseases which are not specifically of surgical interest, with explanations of the specific causes and measures employed in dealing with them.

Considerable space is given to environmental and industrial hazards, and finally the psychogenic factors which may affect the person for good or ill are well presented. Since the author's interest in the subjects presented in this book was stimulated by the necessity of preparing material for the instruction of medical students the various chapters are in the style of didactic lectures. This, however, does not detract from their value because each subject is well presented in good literary style. Time devoted to reading this book will be well spent by medical students and those physicians who have not kept up with the scientific details of the subjects presented.

A Practice of Orthopaedic Surgery. T. P. McMurray. 471 pp. Baltimore: William Wood & Company, 1937. $5.00.

This is a concise, well-written textbook of orthopedic surgery, sufficiently illustrated for practical purposes.

The author works in Liverpool and therefore represents the school that Sir Robert Jones dominated. Since the practice of this school is closely connected with that in the United States and Canada, the book ought to have a definite appeal to students and practitioners in this country.

It is a very well-proportioned book, giving a fair share of space to the major subjects of tuberculosis, infantile paralysis and scoliosis. It also — and the reviewer believes this is a good feature — deals sufficiently with the minor subjects, such as tennis elbow, slipping tendons and flat feet.

There is no pretense of being complete in the technic of bone surgery and the different types of operative procedures. In the preface the author states that these features have been omitted, but he gives the essentials of treatment in a concise way.

Medicine for Nurses. C. Bruce Perry. 210 pp. Baltimore: William Wood & Company, 1938. $2.00.

An English book, better than the average of its type, has been written by the professor of medicine of the University of Bristol Faculty of Medicine. The book is sound but of local interest, and would not be useful in American hospitals.

Fractures and Dislocations for Practitioners. Edwin O. Geckeler. 252 pp. Baltimore: William Wood & Company, 1937. $4.00.

The author has succeeded in keeping this small book simple, being content to describe one method of treatment that can be used by the so-called general man. He advocates the use of plaster bandages and simple apparatus. To the reviewer it seems to be a valuable book for reference and serves its purpose.

Eat and Keep Fit. Jacob Buckstein. 128 pp. New York: Emerson Books, Inc., 1938. $1.00.

This book, by a recognized authority in the subjects treated, presents in the early chapters the essential dietary principles concerned in the maintenance of health and the prevention of disease.

In order to make plain the problems of nutrition the author gives a description of the structure, composition and chemical elements of the cells of the animal organism with explanations of metabolism and the production of energy incident to the functional activities of the body as a whole.

After exposing the crude and unscientific claims of food faddists, the practical and scientific principles applicable to the maintenance of normal weight of the human beings are clearly set forth.

The fourteen-day reducing diet is an important feature of the book which may be read by many doctors and patients to advantage, and the food value charts furnish useful information for dietitians.

The book is well written, and the facts are presented in an interesting manner.